

**UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF PENNSYLVANIA**

IN RE: NATIONAL FOOTBALL LEAGUE:
PLAYERS' CONCUSSION
INJURY LITIGATION

No. 2:12-md-02323-AB

MDL No. 2323

Kevin Turner and Shawn Wooden,
*on behalf of themselves and
others similarly situated,*
Plaintiffs,

CIVIL ACTION NO: 14-cv-0029

v.

National Football League and
NFL Properties LLC,
successor-in-interest to
NFL Properties, Inc.,
Defendants.

Hon. Anita B. Brody

THIS DOCUMENT RELATES TO:
ALL ACTIONS

**DECLARATION OF
KRISTINE YAFFE, M.D.**

I, KRISTINE YAFFE, M.D., hereby declare as follows:

1. If called as a witness, I could and would testify competently to the facts herein.

I. Qualifications

2. As discussed in more detail below, I am a Professor in the Departments of Psychiatry, Neurology and Epidemiology at the University of California San Francisco ("UCSF"). In addition to my teaching responsibilities, I also am a researcher, epidemiologist, and practicing neurologist who treats patients suffering from neurodegenerative diseases. My complete *curriculum vitae* is attached as Exhibit A, and I highlight here some of my qualifications, experience, and research relevant to the

opinions I express below. I submit this declaration in support of the NFL Parties' Memorandum of Law in Support of Final Approval of the Class Action Settlement Agreement and in Response to Objections.

3. I obtained a Bachelor of Science in Biology and Psychology from Yale University in 1985. I obtained a Doctor of Medicine from the University of Pennsylvania in 1989. I completed two residency trainings in Neurology and Psychiatry at UCSF in 1995. I completed fellowships in Clinical Epidemiology at UCSF in 1997 and Geriatric Psychiatry at San Francisco Veteran's Affairs Medical Center ("SF VA") in 1997. I am licensed by the National Board of Medical Examiners (California Medical License # G069893).

4. I have been on the faculty at UCSF since 1997. In addition to my responsibilities as a professor, as a clinical neurologist and geropsychiatrist, I regularly treat patients who are suffering from or dealing with Alzheimer's disease ("AD"), Parkinson's disease ("PD"), Amyotrophic Lateral Sclerosis ("ALS"), and dementia at the UCSF Medical Center. I have been treating patients with these conditions since I received my Doctor of Medicine in 1989. One of my areas of specialization and expertise includes the assessment, evaluation, and treatment of patients with neurodegenerative diseases.

5. As part of my research, I focus on predictors of cognitive decline and dementia in older adults, specifically identifying novel strategies to prevent cognitive decline. This research also includes a focus on the association between traumatic brain injury ("TBI") and dementia.

6. I also serve as (i) the director of the UCSF Dementia Epidemiology Research Group, which conducts research relating to cognitive function and dementia in aging populations throughout the United States; (ii) the Chief of Geriatric Psychiatry and Directory of the Memory Disorders Clinic at the SF VA; (iii) the principal investigator of the data core for the Alzheimer's Disease Research Center at

UCSF; (iv) the principal investigator for the San Francisco Veteran Affairs Mental Illness Research, Educational and Clinical Center (MIRECC) Fellowship, which trains fellows in neurology, psychiatry and geropsychiatry; (v) the Roy and Marie Scola Endowed Chair in Psychiatry; and (vi) the Vice Chair of clinical and translational research at UCSF.

7. I am the principal investigator of seven grants from the National Institutes of Health (“NIH”), including one investigating cognitive decline among patients with chronic renal insufficiency, one investigating how sleep dysfunction is associated with cognitive impairment, one investigating how depression in late life is associated with dementia and other outcomes, one focusing on trying to better characterize successful cognitive aging, and one investigating predictors and outcomes of cognitive trajectories in older patients. I am also the principal investigator of three grants focusing on TBI, funded primarily by the Department of Defense.

8. I have published extensively in the areas of cognitive decline and TBI, including publishing over 300 peer reviewed articles in journals such as the Journal of the American Medical Association, the British Medical Journal, the New England Journal of Medicine, and the Archives of General Psychiatry and Annals of Neurology.

9. In my role as a consulting expert for the NFL Parties in this litigation, I am being compensated for my time through UCSF at my standard hourly rate.

II. Assignment

10. I have reviewed the Settlement Agreement and Exhibits 1, 2, and 3 to the Settlement Agreement—the Injury Definitions, the Test Battery and Specific Impairment Criteria, and the Monetary Award Grid. I also have reviewed certain objections to the proposed Settlement relating to medical or scientific issues, including the supporting declarations of Dr. Robert Stern, Dr. Samuel Gandy, and Drs. Brent Masel and Gregory O’Shanick.

11. I have been asked to provide medical and scientific testimony regarding the challenges of proving a causal link between concussions or subconcussive hits, *i.e.*, mild repetitive traumatic brain injury, and cognitive or other impairments, both as a matter of general causation—whether mild repetitive traumatic brain injury causes cognitive or other impairment on a population level—and as a matter of specific causation—whether a specific individual player could establish that his specific cognitive or other impairment was caused by mild traumatic brain injury experienced during his career in the NFL. I also have been asked to provide medical and scientific testimony relating to certain objections, namely, (i) the objections regarding CTE; (ii) the objections regarding the offsets in the Agreement for stroke and TBI; and (iii) the objections that the Settlement does not compensate for Multiple Sclerosis. All of the opinions expressed herein are offered to a reasonable degree of medical certainty and are consistent with opinions that I would offer in my clinical practice.

III. Summary of Opinions

12. As discussed in detail below, I offer the following opinions:

13. First, the science regarding the association between TBI of all severities and the Qualifying Diagnoses in the Settlement Agreement other than “Death with CTE”, *i.e.*, dementia, Alzheimer’s disease, Parkinson’s disease, and ALS, continues to evolve and improve. Based on the science as it stands today, there is an emerging consensus of an association between TBI of all severities, including mild repetitive TBI, and the Qualifying Diagnoses.

14. Second, the nature of the association between TBI of all severities, including mild repetitive TBI, and the Qualifying Diagnoses has not been determined to be a *causal* one.

15. Third, numerous other risk factors have been identified as increasing the risk of developing these same neurodegenerative syndromes, which are independent of TBIs. Such risk factors include, but are not limited to, age, education,

family history, cardiovascular disease, stroke, diabetes, high blood pressure, obesity, and sleep conditions. This means that, in addition to the possible role of mild TBI, there are numerous other risk factors that could be equally as associated with the Qualifying Diagnoses.

16. Fourth, in light of the uncertainty surrounding the causal relationship between mild repetitive TBI and the Qualifying Diagnoses, establishing to a reasonable degree of medical certainty that a mild TBI, or series of mild TBIs, from NFL play *caused* a developed condition would be exceedingly difficult in my opinion—both on a general population basis and even more so on a specific basis with respect to an individual player.

17. Fifth, this difficulty would be even more pronounced in seeking to establish to a reasonable degree of medical certainty that a mild TBI, or series of mild TBIs, from NFL play *caused* CTE. The science regarding CTE is still in its infancy and the causes of CTE are unknown.

18. Sixth, even if one assumes that there is a causal relationship between mild repetitive TBI and CTE, the Settlement, by compensating for dementia and other conditions that have been identified as comorbid, *i.e.*, co-occurring, with CTE, compensates most of the key alleged impairments associated with CTE.

19. Seventh, the stroke and TBI offsets in the Settlement are scientifically justified.

20. Eighth, there is no scientifically established association between Multiple Sclerosis and TBIs, let alone any evidence of causation.

21. Ninth, a doctor cannot—based solely on the patient’s account of earlier symptoms—retrospectively diagnose with any certainty when exactly a patient’s condition first manifested.

IV. Opinions

A. The Challenges of Proving Causation

22. I understand that if these cases moved forward through litigation, the plaintiffs would be required to prove that the head trauma that they experienced in the NFL caused their alleged injuries. As I explain in more detail below, it is my opinion that it would be exceedingly difficult for a retired player to establish as a matter of science and medicine that mild repetitive TBI *caused* his cognitive or other impairment. I describe below some of the challenges that plaintiffs would face in establishing a causal association between mild repetitive TBI and the various qualifying diagnoses agreed to in the Settlement. In order to do so, I first provide background on epidemiology generally. I then describe the state of science with respect to TBI and the scientific community's current understanding of the association between mild repetitive TBI and the various Qualifying Diagnoses. More specifically, I explain that based on the current state of the science, the medical and scientific communities have not yet determined that mild repetitive TBI causes any of the Qualifying Diagnoses. Finally, I address the fact that many other risk factors, independent of TBI, are risk factors for the Qualifying Diagnoses. In fact, it is scientifically established that age is the most significant risk factor for these Qualifying Diagnoses, which are all age-related conditions.

i. Understanding the Difference Between Association and Cause

23. Epidemiology is the study of the distribution and determinants of health-related states or events, including disease, and the application of this study to the control of diseases and other health problems. *See* World Health Organization, <http://www.who.int/topics/epidemiology/en/> (last visited October 30, 2014).

24. One aspect of epidemiology involves researching and attempting to understand the association between an event or agent and a disease. An agent or event that makes it more likely on a population basis that people will develop a disease or

condition is considered a risk factor. Some risk factors have a causal association with a disease. For example, it is now well-accepted that human papillomavirus (HPV), which is a risk factor for developing cervical cancer, in fact, causes cervical cancer on a population basis. Other events or agents may only be risk factors for a disease, and may not have a causal association. For example, physical inactivity is a risk factor for cardiovascular disease, but physical inactivity does not directly cause cardiovascular disease from a scientific standpoint.

25. Using the examples above, the scientific community sufficiently understands the association between HPV and cervical cancer to conclude that HPV is a cause of cervical cancer. In other words, if a woman has cervical cancer it is exceedingly likely that she also has or had HPV. On the other hand, physical inactivity is not considered to be a cause of cardiovascular disease. If an individual develops cardiovascular disease, it may have had little or nothing to do with that individual's physical inactivity. More likely, the physical inactivity is also linked to other risk factors such as higher weight, high blood pressure, dyslipidemia, smoking and poor health that in turn are more directly connected to cardiovascular disease. Nonetheless, physical inactivity is a risk factor for developing cardiovascular disease because it increases the likelihood on a population basis that people will develop cardiovascular disease.

26. In both circumstances, the event or agent increases the likelihood that the patient has developed or will develop the disease. That likelihood, or association, is described as relative risk ("RR"). RRs are assessed on a population basis. A RR explains the increased (or decreased) probability that a member of an exposed group will develop a disease relative to the probability that a member of an unexposed group will develop the same disease.

27. A RR of 1.0 means that the agent has no effect. In other words, if the RR is 1.0 it means that the unexposed group has the exact same probability of developing the relevant condition as the exposed group. A RR of 2.0 indicates that the

risk is doubled for the exposed group. The stronger the association, the more likely it is that a causal component exists. A RR of 5.0 is more likely to be a causal association than a RR of 2.0. In any study, the statistical significance of the association, which is measured through confidence intervals, is also a key consideration. Epidemiologists require a 95 percent confidence interval or “p value” in order to accept that the association is likely to be real and less likely to be based on chance.

28. The RR is far from the only factor that determines or helps assess whether the association is a causal one. Before the scientific community will accept that an event or agent is a cause of a disease, a number of additional factors must be satisfied. All of these factors also must be judged in light of the quality and quantity of studies examining the association between the agent or event and the disease. The well-accepted factors that must be considered before a causal association can be established include: strength of association, consistency, specificity, temporality, biological gradient, biological plausibility, experimental evidence and analogy. I will discuss each in turn.

29. “Coherence” or “consistency” examines whether external trends over time are consistent with the predicted association. For example, for cigarette smoking, a marked increase in lung cancer death rates followed the increase in sales of cigarettes. Had the increase in lung cancer deaths followed a decrease in cigarette sales, the inconsistency would have to be explained.

30. “Specificity” refers to the idea that the factor must influence specifically a particular outcome or population.

31. “Temporality” refers to the fact that a factor must precede an outcome that it is supposed to affect.

32. “Biological gradient” refers to the notion that an outcome increases monotonically with increasing the dose of exposure or according to a function predicted by a substantive theory.

33. “Biological plausibility” refers to whether the observed association is biologically possible given studies surrounding the specified agent and the studied disease.

34. “Experimental evidence” refers to the notion that causation is more likely if evidence is based on randomized experiments.

35. “Analogy” focuses on whether other similar agents (for instance a drug in the same class as the agent being examined) could equally induce the disease (i.e. whether an effect has already been shown for analogous exposures and outcomes).

36. It is only after these factors are thoroughly tested through prospective, controlled studies that it may be possible to conclude, as a matter of science and medicine, that an event or agent is actually a *cause* of a disease, rather than simply a risk factor for that disease. See *Gulf War and Health: Long-Term Consequences of Traumatic Brain Injury*, Vol. 7, 106 (2008) (“Gulf War and Health”).

37. When such criteria have not been established or studied, the scientific community will not be able to determine that the agent is the cause of the disease. Rather, it is only possible to say that the agent or event has an *association* with the disease. For example, carrying matches has a RR of developing lung cancer of at least 1.7. In other words, individuals who carry matches are 70% more likely to develop lung cancer than individuals who do not carry matches. Logically, in that instance, we do not need to do additional tests to understand that carrying matches is not a cause of lung cancer, but is merely associated with another cause of lung cancer, smoking. In other, less obvious cases, the exact nature of the association may be less apparent.

38. Understanding the importance of testing for these various factors, through prospective, controlled studies, helps put into perspective the state of science regarding mild repetitive TBI and its association with both the Qualifying Diagnoses and CTE. In the case of TBI of all severities, while the emerging science believes that TBI

of all severities is a risk factor for developing the Qualifying Diagnoses, it is less clear whether the risk factor has a causal association with any of the Qualifying Diagnoses.

ii. The Association Between TBI and the Qualifying Diagnoses

39. I will now provide my opinions as to the state of the science regarding TBI and the Qualifying Diagnoses. The specific issue I address is whether, as a matter of science and medicine, mild repetitive TBI has a causal association with the Qualifying Diagnoses in the Settlement. It is my belief—and no scientific study says or demonstrates otherwise—that based on the current state of the science, the association between mild repetitive TBI and the qualifying diagnoses is not clear. And even if mild repetitive TBI emerges as a *risk factor* for developing the Qualifying Diagnoses, it is unclear whether this association is a causal one. Because of this, it is also my opinion that, given the state of the science, plaintiffs in this litigation would have an exceedingly difficult time establishing from a scientific standpoint that the mild repetitive TBI, *i.e.*, concussions or subconcussive hits, experienced during their NFL careers *caused* any of the Qualifying Diagnoses on a population basis (general causation) or in any individual instance (specific causation).

40. In order to put my opinions in perspective, it is first important to understand the nature of scientific knowledge and study in this area and the limitations of the studies that have been conducted.

41. The first limitation involves the severity of TBI that traditionally has been studied. In general, the scientific community categorizes TBI into three categories: severe, moderate, and mild. More recently, the scientific community has sought to establish a more detailed definition of TBI.¹ But while a uniform definition is

¹ In the past, the Glasgow Coma Scale was the most used tool to assess the severity of a TBI. Under the Glasgow Coma Scale, patients were evaluated regarding eye response, verbal response, and motor response following the TBI. The cumulative score in these areas led to the classification of the TBI. More recently, the scientific community has sought to establish a more detailed definition of TBI. Thus, the

being established, different criteria have been used to assess severity across these studies, and often TBI in studies was measured solely through the subject's own self-reporting, resulting in data of uncertain reliability. This means that when a study finds that a TBI is a risk factor for or associated with a certain condition, it is often unclear whether the study means severe TBI, moderate TBI, mild TBI, or repetitive TBI—or any mix of these combinations. Because different levels of severity in a TBI have been shown to change the resulting effects, the studies must be reviewed carefully to understand what severity of TBI was being studied. This makes reaching scientific conclusions in this area even more difficult.

42. Moderate and severe TBI have been studied significantly more than mild TBI. Based on the science to date, including my own, it is my opinion that (i) moderate and severe TBI have a strong association with late-life dementia and the other Qualifying Diagnoses, (ii) mild repetitive TBI may have an association with late-life dementia and the other qualifying conditions, but at a later age than moderate or severe TBI, and (iii) *none* of these associations have yet to be established as causal ones.

43. Moreover, the studies that have occurred have not established a scientific consensus in this area. While the majority of studies suggest the associations I describe above, the overall results are mixed. For instance, while many studies have found that moderate to severe TBI have an association with the Qualifying Diagnoses under the Settlement Agreement with a RR of between 1.5 and 2.0, some studies have *not* found an association between TBI of any severity and the qualifying conditions. *See, e.g.,* K.M. Mehta, *Head Trauma and Risk of Dementia and Alzheimer's Disease*, 53 *Neurology* 1959, 1959-62 (1999). Other studies have found that TBI is a risk factor for dementia, but *only* when the severity of the head injury is above a certain threshold

scientific community currently accounts for loss of consciousness and post-traumatic amnesia, in addition to the Glasgow Coma Scale, in assessing severity of TBI.

(moderate or severe, but not mild). See Kevin Guskiewicz, et al., *Association between Recurrent Concussion and Late-Life Cognitive Impairment in Retired Professional Football Players*, Vol 57 JNS 718, 720 (October 2005) (“Guskiewicz Study”); see also B.L. Plassman, et al., *Documented Head Injury in Early Adulthood and Risk of Alzheimer’s Disease and other Dementias*, 55 JNS 1158, 1158 (2000) (“Plassman Study”) (finding that “the association between antecedent head injury and AD is inconsistent.”).

44. My own study, published in October 27, 2014, along with my colleagues, reflected the evolving nature of the field and concluded in part that “mild TBI² sustained at 65 years or older or moderate to severe TBI sustained at 55 years or older may significantly increase the risk of developing dementia. . . . The effect of mild TBI sustained in middle age or earlier deserves further study during a longer period of follow-up.” See R. Gardner, et al. *Dementia Risk After Traumatic Brain Injury vs Nonbrain Trauma*, JAMA Neurology (October 2014) (“Gardner Study”).

45. With respect to mild repetitive TBI specifically, which I understand to be the focus of causation issues in this litigation, the studies in this area have been more recent and are still evolving. These studies also have some additional limitations, including lack of a representative sample, selection bias, lack of control for potential confounding factors, self-reports of exposure and health outcomes, and outcome misclassification. See Gulf War and Health at 113; see also Guskiewicz Study.

² In this study, we used the definition of mild TBI set forth in the National Center for Injury Prevention’s report to Congress on mild TBI. See *Steps to Prevent a Serious Public Health Problem*, Report to Congress on Mild Traumatic Brain Injury in the United States (Sept. 2003), available at <http://www.cdc.gov/ncipc/pub-res/mtbi/mtbireport.pdf> (defining mild TBI as “[a]ny period of observed or self-reported: transient confusion, disorientation, or impaired consciousness; dysfunction of memory around the time of injury; or loss of consciousness lasting less than 30 minutes.” *Id.* at 2.

46. Some studies, such as the Guskiewicz Study, openly acknowledge the limitations of the current scientific data regarding the association between mild repetitive TBI and the qualifying conditions, and suggest that though a history of recurrent concussions *may* be a risk factor for mild to severe cognitive impairment, future prospective studies implementing genetic testing and more rigorous established diagnostic criterion are necessary to clarify the causal effects of TBI, and separate its influence from other confounding factors. *Id.* at 723; *see also* Plassman Study at 1158 (finding that moderate and severe head injuries in young men may be associated with increased risk of AD and other dementias in late life, however the possibility that other unmeasured factors were influencing the association was not (and could not be) excluded). These studies reinforce the importance of evaluating all the specified criteria discussed above regarding causation; because mild repetitive TBI's association to the Qualifying Diagnoses has not been thoroughly tested, no assumptions as to whether the association is causal can be made.

47. In sum, the science regarding the association between TBI of all severities and the Qualifying Diagnoses continues to evolve and improve. This is a critical area of continued research. However, based on the science as it stands today, it is my opinion that while there is some association between mild repetitive TBI and the Qualifying Diagnoses, the scientific community is unable to draw a causal link between mild repetitive TBI and the qualifying diagnoses. *See Gulf War and Health* at 114.

48. While my discussion thus far has focused on the association between TBI and the qualifying conditions, it also important to note that numerous other risk factors have been identified as increasing the risk of developing these same neurodegenerative syndromes, independent of TBIs. This means that there are numerous other risk factors that could be equally as likely to have caused the qualifying diagnoses. Such risk factors include, but are not limited to, age, education, family history,

cardiovascular disease, stroke, diabetes, high blood pressure, obesity, and sleep conditions.

49. Moreover, these factors often appear in aggregate and can co-occur. In other words, in many cases, the risk factors individually may not be a high risk factor for developing a certain condition, but when aggregated together could have a high risk factor for that condition. This makes the ability to prove, as a matter of science, that a single event was a cause or a contributing cause of a condition all the more difficult.

50. The difficulty of scientific proof is exponentially increased with age-related diseases, such as all of the Qualifying Diagnoses in the Settlement: Alzheimer's, Parkinson's, ALS, and other forms of dementia, *e.g.*, frontotemporal dementia. It is scientifically established that age is the most significant risk factor for these Qualifying Diagnoses. As such, as subjects increase in age, it is even harder to determine whether the cause of the Qualifying Diagnosis was the mild repetitive TBI, age, or any other risk factor other than TBI (assuming that mild repetitive TBI is even a risk factor), or some combination. What can be said with assurance is that, statistically, it is *more likely* that age was the main risk factor than any of the other identified risk factors, including mild repetitive TBI.

51. The complexities discussed herein are articulated not to complicate the issue, but rather to explain the numerous challenges to establishing that, even on a broad population level, single or repetitive or mild TBIs resulting from NFL football are the cause of—or even one cause of—an individual's development of a neurodegenerative disease. We currently know that moderate to severe TBI in some way increases an individual's risk of developing certain neurodegenerative diseases. And with respect to mild repetitive TBI, we know even less: it likely increases an individual's risk of developing certain late-life neurodegenerative diseases, but we as a scientific community simply cannot say that mild repetitive TBI *causes* any developed syndrome. As with the matches and lung cancer example, the increased risk that we currently see in these studies

might be established due to any number of associated factors (*e.g.*, individuals who experience mild repetitive TBI might have a predisposition—genetic or otherwise—towards aggressive behavior or risk taking, which itself may actually be a risk factor for or a cause of developing a neurodegenerative disease, just as smoking, not carrying matches, is the actual cause of developing lung cancer).

52. On an individual level, establishing causation between mild repetitive TBI and neurodegenerative syndromes is even more difficult. This is logical. Just as on a broad population level it is very difficult and not yet scientifically possible to separate out whether TBI is a cause—or even one cause—of the identified neurodegenerative diseases, on an individual level, even more factors come into play. Each individual player's medical and social history would need to be understood in full detail, but one can imagine many players having many of the significant risk factors for developing a certain condition aside from the prior TBI exposure in NFL football (again, even assuming mild repetitive TBI is a risk factor for the qualifying conditions). Those more significant or similar magnitude risk factors might include age, a specific genetic profile or gene, obesity, cardiovascular disease, depression, post-traumatic stress disorder, or substance abuse. The qualifying conditions have been studied for many years, across many patients, and the etiologies are complex and still not completely understood. There is no simple answer in any individual case.

53. In light of the uncertainty surrounding the causal relationship between mild repetitive TBI and the Qualifying Diagnoses, it is my belief that establishing to a reasonable degree of medical certainty that a mild TBI, or series of mild TBIs, from NFL play *caused* a developed condition would be exceedingly difficult—both on a general population basis and even more so on a specific basis with respect to an individual player.

iii. The Association Between TBI and Chronic Traumatic Encephalopathy

54. I have reviewed the objections regarding CTE, including the medical declarations in support of those objections. I understand that certain objectors criticize the fact that CTE allegedly is not compensated under the settlement after July 7, 2014. I will now describe some of the challenges retired players would have in establishing that mild repetitive TBI causes CTE, which also overlap with the challenges described above in establishing such an association with respect to the Qualifying Diagnoses more generally. In my professional opinion, the objections do not account for the early state of science regarding CTE, which would make it even more difficult to establish causation between mild repetitive TBI and CTE.

55. Currently, the diagnosis of CTE is based solely on a neuropathological diagnosis that, as of today, can only be made post-mortem. This simply means that a neuropathologist must physically look at an individual's brain post-mortem, determine if something called the "tau protein" is present and in a particular pattern, and then diagnose the deceased individual with CTE.

56. Moreover, the science surrounding CTE is in its infancy. The science that does exist is recent, preliminary, and evolving. In my experience, many neurologists are not even aware of CTE. And there is much to be learned.

57. In fact, two highly respected, non-biased institutes—the National Institutes of Health and the Institute of Medicine—have issued recent consensus statements identifying important gaps in the medical and scientific communities' understanding of CTE and have identified important questions regarding CTE that need to be answered. In doing so, these leading institutions have explained the need for additional—and more comprehensive and controlled—clinical testing and research into CTE before we as a scientific community can make any assumptions about the causality of CTE, or establish its symptomatic profile. *See Sports Related Concussions in Youth:*

Improving the Science, Changing the Culture, Institute of Medicine, (Oct. 30, 2013), available at http://www.iom.edu/Reports/2013/Sports-Related-Concussions-in-Youth-Improving-the-Science-Changing-the-Culture/Report_Brief103013.aspx (“[I]t remains unclear whether repetitive head impacts and multiple concussions sustained in youth lead to long-term neurodegenerative diseases, such as chronic traumatic encephalopathy.”); *Report on the Neuropathology of Chronic Traumatic Encephalopathy Workshop*, National Institutes of Health, (Dec. 5-6, 2012), available at http://www.ninds.nih.gov/news_and_events/proceedings/201212_CTE_workshop_report.htm (listing various outstanding questions regarding CTE).

58. I agree with the opinions expressed by these well-respected institutes. More CTE-related research, especially, as described in detail below, research of higher significance on the epidemiological pyramid, is required to advance the scientific community’s understanding of CTE. I will now address the nature of the current studies on CTE, and what are, in my opinion, the severe limitations of these studies.

59. In evaluating the state of the science regarding any condition or diseases, clinicians must assess the kinds and number of studies on the topic. There are many different kinds of studies. Some are more reliable, useful, and persuasive than others. The order of such studies (from most reliable, useful, and persuasive to least) is what is known as the “pyramid of studies” or the “quality of evidence” in epidemiology.

60. The top of the pyramid, *i.e.*, the most reliable, useful, and persuasive type of study, is a double-blind, randomized control trial. Such studies are considered the “gold standard” of epidemiological studies to establish causation. In a double-blind, randomized control trial, two groups of individuals are studied prospectively over long periods of time: one group is randomly assigned to the agent or event at issue, the other is not. The researchers are blinded to which individuals are

exposed. These studies are the gold standard because they provide the best controls and limit many potential biases.

61. A second level of study is a prospective study, which does not have blinding or randomization, but assesses patients at their baseline and then follows and continues to assess them over a long period of time on a prospective basis. These studies also are highly reliable, useful, and persuasive.

62. A third level of study is a cross-sectional study, which looks at different groups of individuals that may or may not have the studied disease at one point in time. These kinds of studies can help separate out what other factors might influence the disease aside from the studied agent, but are not as methodologically strong.

63. A fourth level of study is a case control study, which takes two groups, one with the disease and one without, and attempts to establish the differences in exposures between the groups. In such studies, it can be difficult to determine the extent of exposure because the study often relies on retrospective recollections.

64. Finally, the least reliable, useful, and persuasive type of study is a case report or case series. Such studies are still important to help further the scientific community's understanding of a particular issue, but, like case control studies, case reports are used to help determine hypotheses that must be tested further, rather than to answer scientific uncertainties. Case reports or case series look retrospectively at exposure and outcomes and do not have control groups.

65. With that background, I will now turn to the state of scientific research regarding CTE to explain how difficult it would be for retired players to prove that mild repetitive TBI in the NFL causes CTE, both on a population level and with respect to a specific individual player.

66. There are no published double-blind randomized control trials, prospective studies, cross-sectional studies, or even case control studies regarding CTE. At this juncture, the only available studies are case reports, which, while valuable to the

development of science, make it very difficult to understand a condition or disease in full. And there are only a limited number of these studies to date. Because of the limited research available on CTE, and the lack of long-term, prospective studies, very little is known about CTE. The scientific community does not completely understand the cause or causes of CTE. Nor does the scientific community understand the diagnostic and clinical profile of CTE.

67. Because the scientific community is in agreement that CTE is not yet understood, it logically follows that any causal assumptions made as to concussive or sub-concussive brain impacts relating to CTE are also premature. The same is true of the diagnostic profile of CTE. Any assumptions regarding the symptoms that constitute the diagnostic profile of CTE are yet to be determined.

68. It is helpful to compare our understanding of CTE to our understanding of Alzheimer's disease. Alzheimer's disease has been studied in *millions* of individuals, over *thousands* of clinical trials over the past fifty years. And many of these studies have been long-term, prospective and highly controlled. As for CTE, less than *200* brains diagnosed with CTE have been examined, and there have been no long-term, prospective, *or* highly-controlled studies. The limited studies that have taken place have been retrospective, and, therefore, have many limitations, including, but not limited to, limited sample size, referral bias in the participating sample, lack of proper controls, and information bias.

69. Dr. Robert Stern is the primary expert who filed a declaration in support of the objections. I have reviewed that declaration in which Dr. Stern describes the alleged clinical and diagnostic profile of CTE. Dr. Stern's research, which is conducted with other doctors at Boston University, including Dr. Ann McKee, constitutes important research in the field of CTE at this time. However, Dr. Stern's primary study regarding CTE, see Ann McKee, Robert Stern, et al., *The spectrum of disease in chronic*

traumatic encephalopathy, 136 Brain 43 (2013) (“McKee Study”), is a case report and therefore suffers from all of the limitations of these kinds of studies described above.

70. In the McKee Study, the authors began by reviewing the pathology of 85 brains. The 85 brains included former athletes, military veterans, and civilians with a history of repetitive mild TBI. *See* McKee Study at 55. The authors then gathered information about half of the subjects’ lives through retrospective interviews of the subjects’ next of kin. Approximately half of the subjects were eliminated from this key portion of the study for various reasons, possibly including the authors’ inability to contact the next of kin. A medical record review of each of the 45 subjects was also conducted. *Id.* In the study, the authors concluded that CTE is a progressive disease in four stages—CTE I through IV—and attempted to ascribe symptoms to each stage based on the retrospective calls to family members of the decedents. *Id.* at 55-63. Generally, the authors concluded that mood and behavioral symptoms, in addition to some cognitive impairment, are associated with CTE I and II, and that individuals with CTE III and CTE IV progress to dementia. *Id.* The authors also found that many of the subjects in the study had CTE and co-morbid, *i.e.*, co-occurring disease, which included Alzheimer’s, Parkinson’s, ALS, and frontotemporal dementia. *Id.* at 59.

71. Studies such as this one should be lauded and praised for pioneering the science of CTE. However, as discussed in detail, case reports are inherently limited because of their intrinsic biases and lack of controls. The McKee Study contains many such potential biases. First, the sample involved in the McKee Study was self-selected: the subjects donated their brains to Boston University likely *because* they suspected they were suffering from CTE. And family members agreed to allow for a pathological review of the subjects’ brains likely for the same reason. This is known as referral bias. Second, the study lacked proper control subjects to compare against the participating subjects. Third, retrospectively interviewing family members who suspected their deceased relatives had CTE and who were being asked to recall

alleged symptoms that occurred months or years before the interviews—without any medical training in assessing symptoms—is filled with limitations. This is known as information or recall bias.

72. A second study conducted by Dr. Stern suffers from the same limitations. In that study, Dr. Stern and his colleagues used the same methodology as the McKee Study to assess the clinical symptoms allegedly associated with individuals diagnosed post-mortem with CTE. Robert Stern, et al., *Clinical presentation of chronic traumatic encephalopathy*, 81 *Neurology* 1122 (2013) (“Stern Study”). In fact, the Stern Study largely relied on the same subjects as the McKee Study for the diagnosis of CTE. Of the subjects whose family members were contacted in phone calls—36 in total—28 were included in the McKee Study, and 8 were new. *Id.* In addition, a number of potential subjects were excluded from the group of overall subjects whose brains were examined, including individuals with the presence of comorbid disease. Dr. Stern and his colleagues conclude in the study that “there may be 2 different clinical presentations of CTE, with one initially exhibiting behavioral or mood changes, and the other initially exhibiting cognitive impairment.” *Id.* at 1124. Out of the 33 symptomatic patients (3 were asymptomatic, including 2 former professional football players) whose symptoms were collected through the retrospective calls, almost 85% experienced memory impairment, almost 79% experienced executive dysfunction, almost 73% experienced attention and concentration difficulties, almost 58% experienced language impairment, and 54.5% experienced visuospatial difficulties. *Id.* at 1126-27.

73. As with the previous study, this study is a case series and as such has intrinsic limitations. Dr. Stern and his colleagues warn about some of these limitations of their study: “Although these findings are based on the largest cohort of subjects with neuropathologically confirmed CTE without comorbidities studied to date, interpretation and generalizability of these results are limited by several factors.” *Id.* at 1127. Those factors include: the overall sample size being small, which the authors note

means that “caution should be taken when generalizing these results to the larger population of athletes or to the overall clinical presentation of CTE”, inherent selection bias given the self-selection of participants who donated their brains, the further reduction of the sample by eliminating individuals with comorbid pathology, the “potential for reduced reliability and validity of retrospective reports from family members after the death of a loved one,” and the lack of a comparison group of former athletes without CTE. *Id.* Dr. Stern and his colleagues also concluded that “[f]uture research is needed to clarify the clinical presentation of CTE.” *Id.*

74. Given the types of limitations described by Dr. Stern in the Stern Study, the scientific community has used case report studies—such as the McKee Study and the Stern Study—only to consider hypotheses to be further tested, and not to draw any conclusions as to causality or diagnostic profiles. Just as with the concern of incorrectly attributing the association between matches and lung cancer to causality, the symptoms reported by family members as prevalent in the football players could arise from other conditions, entirely unrelated to CTE. Until further prospective studies examine these associations more closely, in my opinion, it appears that the scientific community agrees that it is premature to accept the McKee Study’s conclusions relating to CTE as anything more than hypotheses. *See, e.g.,* Robert Stern, Ann McKee, et al., *Chronic Traumatic Encephalopathy: Where are We and Where are We Going?*, 13 Curr. Neurol. Neurosci. Rep. 407 (2013) (“CTE research is just beginning,” and requires more prospective, longitudinal epidemiological studies to “better understand CTE.”); Christine Baugh, et. al, *Chronic Traumatic Encephalopathy: Neurodegeneration Following Repetitive Concussive and Subconcussive Brain Trauma*, 6 Brain Imaging & Behavior 244 (2012) (“Research related to CTE is in its infancy. Although the neuropathology of CTE has been elucidated in recent years, important areas of research remain, including investigations of CTE’s epidemiology, specific risk factors (in addition to repetitive brain

trauma exposure), underlying disease mechanism, and the ability to diagnose CTE during life.”).

75. Despite these known limitations, many Objectors focus on the fact that the Settlement does not compensate mood and behavioral symptoms, standing alone. On this point, Dr. Stern opines in his declaration that it is scientifically established that CTE causes certain mood and behavioral symptoms. As discussed, it is not yet possible to state with any certainty whether CTE causes any mood or behavioral symptoms. In addition, there is the added concern that these neuropsychiatric symptoms are quite common in the general population. Mood and behavioral symptoms have multifactorial—not unitary—causation. This means that it is an accepted fact that mood and behavioral symptoms generally are not caused by one condition alone, but rather are manifested as the result of many risk factors and etiologies aggregated together. For NFL players in particular, a typical retired NFL player might have one of these many risk factors for depression or mood and behavioral issues, such as sleep apnea, family history, higher BMI, drug abuse, or exposure to severe lifestyle changes. All of these factors could be the cause of the symptoms identified by the subjects’ family members, and all of these factors could be entirely unrelated to CTE. Put simply, because mood and behavioral symptoms, such as depression, have many risk factors, these symptoms could be completely unrelated to CTE.

76. Relatedly, I have reviewed objections that claim that CTE causes suicidal tendencies. This same reasoning concerning mood and behavioral symptoms also applies to suicidal tendencies. As an initial matter, there are no scientifically established studies that I am aware of that show a relationship between suicide and contact sports. But even if such a study did exist, the causes of suicide are as complex, multifactorial, and difficult to predict in individual instances as mood and behavioral symptoms are more generally. Just as we cannot yet know whether mood and behavioral

symptoms are caused by CTE, there is no scientific research that I am aware of confirming that there is *any* correlation between suicidal tendencies and CTE.

77. Dr. Stern and Dr. Gandy also suggest that their teams and others in the field will develop highly accurate and clinically accepted—even FDA approved—methods to diagnose CTE in a living individual in the next five to ten years. Objectors use this point to argue that CTE should be compensated under the settlement because clinicians will be able to diagnose CTE in living people. But they may misunderstand what Dr. Stern and Dr. Gandy are suggesting. They are suggesting merely that the scientific community might develop a way to diagnose the presence of tau protein, which signals CTE, through what is known as a PET scan in a living individual. But that does not mean that we will soon understand what causes CTE or the diagnostic profile of CTE (*e.g.*, what symptoms are caused by CTE). It will take many years before science can fully understand these issues. I am hopeful that research in this area will continue to advance the scientific community's understanding of this degenerative condition. By comparison, even with the tremendous research and thousands of clinical studies on millions of individuals over the past fifty years regarding Alzheimer's, there is still uncertainty as to its causes and effects.

78. In sum, the science surrounding CTE is premature, evolving, and does not include any prospective or long-term studies. Because of this, the studies we do have—which are susceptible to potential biases and limitations—are not sufficient to create a credible diagnostic profile for CTE, or to draw any conclusions as to causality. This is particularly true in the context of mood and behavioral symptoms, which are very common in the general population and have numerous causes; attributing these symptoms to CTE in particular, will always be especially difficult.

79. Therefore, based on the state of the science, in my professional opinion, there are significant challenges to establishing that, even on a broad population level, single or repetitive or mild TBIs resulting from NFL football are the cause—or

even one cause—of an individual’s development of CTE. These challenges are even more acute than those faced by players seeking to establish causation between mild repetitive TBI and the Qualifying Diagnoses.

B. The Settlement Provides Compensation For the Key Alleged Features of CTE

80. More importantly, even if one assumes that there is a causal relationship between mild repetitive TBI and CTE, the Settlement compensates the key alleged impairments associated with CTE given what we know about CTE as of today. By compensating for dementia and other conditions that have been identified as comorbid, *i.e.*, co-occurring, with CTE, the vast majority of retired players who manifest symptoms of CTE while living will be compensated according to the studies relied upon by objectors. As discussed below, my opinions are confirmed by the current studies conducted by Dr. Stern and Dr. McKee to date.

81. Objectors allege that there are a litany of mood and behavioral symptoms that are part of the diagnostic and clinical profile of CTE: memory impairment, executive dysfunction, language impairment, visuospatial difficulties, impaired concentration and attention, depression, apathy, irritability, suicidality, headaches, aggressiveness, disinhibition, explosivity, mood instability, gait disturbance, tremors, muscle weakness, and spasticity, a sensitivity to noise, visual impairment, chronic pain, dysnomia, peripheral nerve dysfunction (numbness, burning, and/or tingling), cervical spinal disorders, sleep dysfunction, and somatic disorders. But even within that long list of symptoms are symptoms that are associated with dementia and undisputedly are covered under the Settlement, namely, memory impairment, executive dysfunction, language impairment, visuospatial difficulties, impaired concentration and attention. Based on the McKee Study and the Stern Study, it appears that almost all players with CTE have impairment in these areas, known as cognitive domains, that are associated with dementia (memory and learning, complex attention, executive function,

language, and visuospatial), and that players who progress to CTE III or CTE IV will develop dementia.

82. More specifically, it appears to me that the vast majority of the former professional football players who were examined in the McKee Study would have been compensated under the terms of the settlement. The McKee Study concluded that “thirty-one of the thirty-four former professional American football players had stage III–IV CTE or CTE plus co-morbid disease (89%).” *See* McKee Study at 59. “CTE plus co-morbid disease” refers to players who were diagnosed with CTE and one of the following: Alzheimer’s disease, Parkinson’s disease, ALS, or frontotemporal dementia. *Id.* The McKee Study further reported that individuals with CTE III or CTE IV had symptoms consistent with dementia. In other words, at stage III, “[t]he most common presenting symptoms were memory loss, executive dysfunction, explosivity and difficulty with attention and concentration.” *Id.* at 56. At stage IV, “[e]xecutive dysfunction and memory loss were the most common symptoms at onset, and all developed severe memory loss with dementia during their course. Most subjects also showed profound loss of attention and concentration, executive dysfunction, language difficulties, explosivity, aggressive tendencies, paranoia, depression, gait and visuospatial difficulties.” *Id.* at 58-59.

83. Based on my review of the injury definitions and test battery agreed to under the Settlement Agreement, and accepting the findings in the McKee Study as accurate, at least 89% of the former NFL players studied by Dr. Stern, Dr. McKee, and their colleagues would have been compensated under the settlement while living. Additionally, it is possible that the remaining three players may have received compensation while living; we simply do not know enough about their symptoms or the severity of their symptoms based on the text of the McKee Study.

84. It is thus my belief that while we do not know enough about CTE to adequately understand its diagnostic profile or causal properties, from the information

we do know, the settlement compensates symptomatic players with CTE by compensating for dementia and other conditions that have been identified as comorbid with CTE.

C. The Stroke Offset is Scientifically Justified

85. I have reviewed objections that argue the stroke offset in the Settlement is not scientifically justified. Objectors argue that stroke is caused by mild repetitive TBI experienced during NFL play and that retired players, therefore, should not be subject to an offset for an event caused by their NFL careers. In my opinion, there is a significant association between stroke and dementia. Moreover, while there is an association between TBI and stroke, that association has been identified with moderate and severe TBI, not mild repetitive TBI. Therefore, in my professional opinion, the stroke offset is justified and reasonable.

86. There is a significant association between stroke and dementia. *See, e.g.,* J.S. Elkins, Kristine Yaffe, et al., *Pre-existing Hypertension and the Impact of Stroke on Cognitive Function*, 58 Ann. Neurol. 68 (2005) (clinical study concluding that pre-existing hypertension resulting from a stroke was a strong predictor of cognitive decline); R. Sahathevan, R., et al., *Dementia, Stroke and Vascular Risk Factors; A Review*, 7 Int. J. Stroke 61 (“There is also substantial evidence that stroke risk factors such as hypertension . . . are independently associated with an increased risk of Alzheimer’s disease, and vascular cognitive impairment. . . . Physicians must be aware that stroke causes dementia.”).

87. In fact, strokes are recognized as the second most common cause of dementia, known in the scientific community as “vascular dementia.” This is a distinct kind of dementia primarily caused by the occurrence of one or more strokes.

88. Objectors argue that even if stroke is causally related to dementia, it is playing football itself that often causes the stroke. In support of this argument,

Objectors cite to a study conducted by Dr. Burke and his colleagues. *See* Burke, J.F., et al., *Traumatic Brain Injury May be an Independent Risk Factor for Stroke*, 81 NEUROLOGY 33 (2013). The Burke study found that subsequent stroke was found in 1.1% of the group of patients with a prior TBI (and in 0.9% of the control group) over a period of 28 months. However, this study naturally focused on moderate to severe TBIs by relying on TBIs that resulted in subjects going to the emergency room at a hospital or were admitted to the hospital on an inpatient basis due to their TBIs. This study thus determined that moderate to severe TBI—not mild TBI—is associated with stroke, independent of other major predictors. A second study cited by objectors for the same purpose, *see* Y. H. Chen, *Patients with Traumatic Brain Injury: Population-Based Study Suggests Increased Risk of Stroke*, 42 Stroke 2733 (2011) (“Chen Study”), also showed an increase risk of stroke following moderate to severe TBIs during the first three months to a year, with a much lower potential association at the five year mark, but—like the Burke Study—the Chen study did not address or draw any conclusions relating to mild repetitive TBI. A third study described by Objectors, although not cited, *see* Chien-Chang Liao, PhD, MPH, *Stroke Risk and Outcomes in Patients with Traumatic Brain Injury: 2 Nationwide Studies*, 89 Mayo Clin. Proc. 163, 163-172 (2014), found that mild TBI was associated with stroke during the 24-month period following the TBI. The first two studies thus do not address the principal theory of Objectors’ complaint—that mild repetitive TBI causes stroke. The final study does not address the theory that mild repetitive TBI causes remote stroke years after NFL play.

D. The Traumatic Brain Injury Offset is Scientifically Justified

89. I have reviewed objections that argue the TBI offset is not scientifically justified. In my opinion, there is a significant association between moderate and severe TBI and the qualifying conditions. In the Settlement Agreement, the offset only applies if there is a severe TBI. Therefore, in my opinion, the TBI offset in the Settlement is scientifically justified.

90. Moderate and severe TBIs have a strong association with dementia, Alzheimer's, and Parkinson's. Scientific research to date shows acute and severe TBIs—including one single acute and severe TBI—are a risk factor for these conditions with a relative risk ranging between 1.5 and 2.0. *See, e.g.,* A. B. Graves, et al., *The Association Between Head Trauma and Alzheimer's Disease*, 131 Am. J. Epidemiol. 491 (1990) (finding through a controlled study a statistically significant association between head trauma and Alzheimer's disease); Y.K. Lee, et al., *Increased Risk of Dementia in Patients with Mild Traumatic Brain Injury: A Nationwide Cohort Study*, 1 PloS One 8 (2013) (finding that TBI is an independent significant risk factor of developing dementia even in the mild type); Anne M. Harris, et al., *Head Injuries and Parkinson's Disease in a Case-Control Study*, 70 Occup. Environ. Med. 839, 840 (2013) (finding that associations between head injuries and Parkinson's were strongest involving concussions or unconsciousness). My personal clinical studies, some of which are discussed herein (*see supra* ¶ 44), also support this position.

IV. There is no Scientifically Established Correlation between Multiple Sclerosis and Traumatic Brain Injuries

91. I have reviewed an objection that argues that multiple sclerosis should be compensated under the Settlement. However, I am not aware of any scientific literature that demonstrates an association between head trauma of any severity and the development of multiple sclerosis. In fact, studies have concluded that there is no association between head trauma and multiple sclerosis. *See, e.g.,* C. C. Pflieger, et al., *Head Injury is not a Risk Factor for Multiple Sclerosis: A Prospective Cohort Study*, National Institutes of Health, (2009), available at <http://www.ncbi.nlm.nih.gov/pubmed/19028828> (concluding that head injury of any severity on either gender does not affect the risk of acquiring multiple sclerosis later in life); M. J. Goldacre, et al., *Risk of Multiple Sclerosis After Head Injury: Record Linkage Study*, 77 J. Neurol Neurosurg Psychiatry 351, 353 (2006) (finding that the RR for

multiple sclerosis from a head injury was 1.1 with a 95% confidence interval, and therefore concluding that there was no significant increase in the risk of multiple sclerosis at either short or long time periods after head injury).

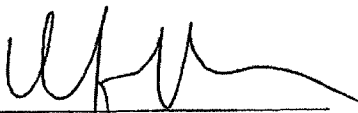
V. It is Difficult For a Clinician to Determine When a Condition Began Without a Contemporaneous Evaluation

92. I understand that certain Objectors complain about Monetary Awards under the Settlement being based in part on the age of diagnosis of a Qualifying Diagnosis. Objectors argue that a retired player should be able to establish through credible evidence that his Qualifying Diagnosis began earlier.

93. In my opinion as a clinician, it is very difficult if not impossible to know when symptoms began, or when a condition actually manifests, without a contemporaneous evaluation. In other words, a doctor cannot—based solely on the patient's account of earlier symptoms—retrospectively diagnose with any certainty when exactly the condition first manifested. The only potential exception would be if a disease were diagnosed at a moderate or severe stage; then, the doctor could potentially say the condition began earlier, but not with any definitive certainty as to even an approximate date.

I declare under penalty of perjury that the foregoing is true and correct.

Dated: San Francisco, CA
November 10, 2014



Kristine Yaffe, M.D.

EXHIBIT A

Curriculum Vitae

KRISTINE YAFFE, M.D.

Roy and Marie Scola Endowed Chair in Psychiatry
Vice Chair of Clinical and Translational Research, Department of Psychiatry
Professor In Residence, Step 6
Departments of Psychiatry, Neurology, and Epidemiology
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EDUCATION

1985	Yale University, B.S. Biology-Psychology
1989	University of Pennsylvania School of Medicine, M.D.
1989-90	Intern in Internal Medicine, Hospital of the University of Pennsylvania
1990-93	Resident in Neurology, University of California, San Francisco
1993-95	Resident in Psychiatry, University of California, San Francisco
1995-97	Fellow in Geriatric Psychiatry, San Francisco VA Medical Center
1995-97	Fellow in Clinical Epidemiology, University of California, San Francisco

Licensure

1990	National Board of Medical Examiners California Medical License # G069893 (expires 7/14)
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Specialty Certification

1995	Board Certified in Neurology, The American Board of Psychiatry and Neurology
1999	Board Certified in Psychiatry, The American Board of Psychiatry and Neurology

CURRENT POSITIONS HELD

University of California, San Francisco
1998- Faculty, Center of Aging

2000- Co-Director, UCSF Memory and Aging Center
 2007- Associate Director, CTSI Mentor Development Program
 2007- Professor In Residence, Departments of Psychiatry, Neurology and Epidemiology and Biostatistics
 2009-2013 Associate Chair of Clinical and Translational Research, Department of Psychiatry
 2009- Roy and Marie Scola Endowed Chair in Psychiatry
 2013- Vice Chair of Clinical and Translational Research, Department of Psychiatry

San Francisco VA Medical Center

1996- Director, Memory Evaluation Clinic
 1997- Chief, Geriatric Psychiatry
 2010- Associate Chief of Service for Research

Kaiser Permanente Division of Research

2008- Adjunct Investigator

PRINCIPAL POSITIONS HELD

1995-97 Attending Physician, Neuropsychiatry Clinic, Outpatient Psychiatry Consultation Service, San Francisco VA Medical Center
 1997-99 Assistant Clinical Professor of Psychiatry, UCSF
 1999-03 Assistant Professor In Residence, Departments of Psychiatry, Neurology and Epidemiology and Biostatistics, UCSF
 2003-07 Associate Professor In Residence, Departments of Psychiatry, Neurology and Epidemiology and Biostatistics, UCSF
 2005-07 Co-Director, Clinical and Translational Sciences Training Program, UCSF

AWARDS AND HONORS

1985 Magna cum laude; Honorary Distinction in Biology
 1988 Alpha Omega Alpha Society, elected junior year
 1989 The Morris Ginsburg Prize for the medical student voted "ideal physician"
 1989 The Henrietta and Jacob Lowenstein Prize in Pediatrics
 1989 American Medical Women's Association Scholastic Achievement Award
 1992 Epilepsy Mini-Fellowship, Bowman Gray School of Medicine
 1994 The West Coast Biological Psychiatry Resident Fellowship
 1994 NIMH Outstanding Resident Award
 1997 Brookdale/NIA Summer Fellowship
 1997 Best Junior Faculty Award: Women's Cognitive Health Conference
 1998 Junior Investigator Award, American Association for Geriatric Psychiatry
 1999 Best Abstract, UCSF Clinical Research Conference
 2001 Paul Beeson Faculty Scholar in Aging Research
 2001 UCSF Alumni-Faculty Association Interdisciplinary Achievement Award
 2002 William B. Abrams Award in Geriatric Clinical Pharmacology
 2004, 2007 Nominated for "Best Mentor" Bay Area Clinical Research Conference
 2005 American Academy of Neurology Research Award in Geriatrics
 2008 American Academy of Neurology Frontiers in Neurology Plenary Talk
 2008 Bay Area Clinical Research Conference "Best Oral Presentation"

2009	Roy and Marie Scola Endowed Chair in Psychiatry
2010	The Royer Award for Academic Excellence in Psychiatry
2010	International Conference on Alzheimer's Disease Plenary Talk
2013	John Mackey Award for Excellence in Dementia Care
2013	UCSF Faculty Research Award in Clinical Science
2013	Invited Scientist G8 Dementia Summit London, United Kingdom
2014	American Association for Geriatric Psychiatry Distinguished Scientist Award
2014	Thomas Reuters World's Most Influential Scientific Minds

Keywords/Areas of Interest

Dementia, cognitive impairment, epidemiology, geriatric psychiatry, prevention

PROFESSIONAL ORGANIZATIONS**Memberships**

1988-	Alpha Omega Alpha Medical Honor Society
1990-	American Academy of Neurology
1993-	American Psychiatry Association
1998-	American Association for Geriatric Psychiatry
2011-	American Neurological Association

Service to Professional Organizations

1999-02	Research Committee, American Association of Geriatric Psychiatry
2001-04	Nominations Committee, American Association of Geriatric Psychiatry
2004-	Abstract Reviewer, American Academy of Neurology Annual Meeting
2007-09	Scientific Planning Committee, International Conference on Alzheimer's Disease and Related Disorders, 11 th and 12 th meetings
2007-	Abstract Reviewer, Alzheimer's Association International Conference on Alzheimer's Disease
2008	Abstract Reviewer, American Geriatrics Society Annual Meeting
2010-11	Co-chair, Alzheimer's Association Research Roundtable in Epidemiology of Dementia
2011-	Clinical Research Committee, American Academy of Neurology
2011-13	Chair, Scientific Planning Committee, 13 th and 14 th International Conference on Alzheimer's Disease and Related Disorders
2012	Abstract Reviewer, American Neurological Association Annual Meeting
2012-	Fellowship Review, American Neurological Association
2012-13	Alzheimer's Association's National Alzheimer's Project Act's (NAPA) Expert Advisory Committee
2012-13	Planning Committee, Institute of Medicine's Cognitive Aging: Translating Science into Prevention and Care Meeting
2013-14	Scientific Planning Committee, 15 th International Conference on Alzheimer's Disease and Related Disorders
2014-	Co-Chair, Committee on the Public Health Dimensions of Cognitive Aging, Institute of Medicine
2014-	Alzheimer's Association Medical & Scientific Advisory Council

SERVICE TO PROFESSIONAL PUBLICATIONS

Updated 10-16-14

Reviewer, American Journal of Medicine
 Reviewer, American Journal of Psychiatry
 Reviewer, Annals of Neurology
 Reviewer, Archives of General Psychiatry
 Reviewer, Archives of Internal Medicine
 Reviewer, Archives of Neurology
 Reviewer, Biological Psychiatry
 Reviewer, Journal of the American Medical Association
 Reviewer, Journal of American Geriatric Society
 Reviewer, Journal of General Internal Medicine
 Reviewer, Lancet
 Reviewer, Neurobiology of Aging
 Reviewer, Neurology
 Reviewer, New England Journal of Medicine

2004-07 Editorial Board, American Journal of Geriatric Psychiatry
 2011- Associate Editor, International Review of Psychiatry
 2012- Associate Editor, Journal of Gerontology: Medical Sciences

GOVERNMENT AND OTHER PROFESSIONAL SERVICE

Grant Review

1998- California Department of Health
 1998-06 The French Foundation for Alzheimer's Research
 1999- The Alzheimer's Association
 2000 NIH Study Section IFCN 8
 2001 Raine Priming Grant
 2001 NIH Special Emphasis Panel 1 IFCN 4
 2002 NIH Special Emphasis Panel Specialized Centers of Research for Women and Gender
 2003 NIH Study Section BBB-4
 2006 NIH Study Section RPHB-G 03
 2008 NIH Study Section AGS
 2008 NIH Toolbox for Assessment of Neurological and Behavioral Function
 2008- AXA Research Fund
 2009 NIH Special Emphasis on Neural and Behavioral Problems of Cognitive Aging
 2009-2014 NIA-N Study Section (standing member)
 2013 Chair, NIDDK Special Emphasis Panel Beta-Cell Function and Cognition
 2013 Review Committee, Brain Canada, Interventions for Prevention of Alzheimer Disease and Related Disorders Multi-Investigator Research Initiative
 2013- Bright Focus Foundation Study Section
 2013- Review Committee, National Alzheimer's Coordinating Center

Other Service

2001 Co-chaired, NIH Healthy Brain Workshop, July 9-10
 2003-10 DSMB, Women's Health Initiative (WHI)
 2004-05 Evaluation Committee, NIH Healthy Brain Workshop
 2006 CDC Prevention of Dementia Working Group

2006 Expert Panel, RAND ACOVE Quality of Care Indicators for the Vulnerable Elderly
 2007 Chair, VA National Subcommittee on Dementia Diagnosis
 2007- Scientific Advisory Committee, AlzForum
 2008- Scientific Advisory Committee, Beeson Scholars in Aging
 2009-10 DSMB (Chair), Pfizer Dimebon Trials
 2009-11 DSMB (Chair), Medivation Dimebon Trials
 2009-11 Advisory Board, Longitudinal Aging Study Amsterdam (LASA)
 2009- DSMB (Chair), Citalopram Treatment for Agitation in Alzheimer Dementia (CITAD)
 2009- National Alzheimer's Coordinating Center (NACC) Scientific Review Committee
 2012- Senate Member, Council of the German Center for Neurodegenerative Diseases
 2012-13 Alzheimer's Disease Diagnostics, National Policy Development Group, Institute for Clinical and Economic Review
 2013- DSMB (Chair), Zinfandel Trial
 2014- External Scientific Advisory Board, Massachusetts Alzheimer's Disease Research Center

UNIVERSITY SERVICE

UCSF Campus-wide

1998 UCSF Vice-Chancellor's Committee on Clinical Research
 1998 Organized and chaired, "Alzheimer Disease and Women: From the Bench to the Bedside and Beyond," with UCSF Center of Excellence on Women's Health
 2002-03 Chancellor's Advisory Panel on Long Term Growth at UCSF
 2005-06 Planning Committee, Clinical and Translational Science Award
 2005-07 Co-Director, Clinical and Translational Sciences Training Program
 2005-09 Advisory Board, Center for Aging in Diverse Communities
 2005-10 Academic Information Systems Board
 2006 Organized and Chaired, "Building a Multidisciplinary and Translational Research Program" conference
 2006-07 Strategic Planning Team: Research Directions
 2007- Associate Director, CTSI Mentor Development Program
 2008- Clinical and Translational Research Pathway Advisory Council, UCSF
 2011-13 Long Range Development Plan Oversight Committee, Research Subcommittee
 2013-14 Stewardship Review Committee, Chair of the Department of Anatomy

UCSF School of Medicine

2000-01 Medical School Curriculum Committee-Brain and Behavior Core
 2004-05 Search Committee, Chair of the Department of Epidemiology
 2006-07 Pathways to Discovery Clinical and Translational Research Committee
 2006-07 Pathways to Discovery Task Force
 2008- Advisory Board, Pathways to Discovery Clinical and Translational Research
 2010-11 Search Committee, Chair of the Department of Psychiatry

UCSF Department of Psychiatry

1995-97 Program for Women Support Committee, UCSF Department of Psychiatry
 1996-97 Committee for Geriatric Psychiatry Education
 2000-01 Search Committee, Schizophrenia Research Faculty

2000-02	Grand Rounds Committee
2001-03	Continuing Medical Education Committee
2002	Friends of Langley Porter Symposium on "Aging with Wit and Wisdom"
2003-06	Co-Director, Clinical Research Training in Psychiatry
2003-06	Grand Rounds Committee
2006-10	Appointments and Promotions Committee
2008	Task Force on Professional and Career Development
2008-09	Chair, Search Committee for Clinical and Translational Research
2009-10	Search Committee for VA-UCSF Research Psychiatrist
2009-12	Search Committee for UCSF Child and Adolescent Psychiatry Researchers
2009-12	Chair, Search Committee for VA-UCSF Geropsychiatry Researcher
2010 -	Chair, Research Retreat
2013-	Chair, Clinical and Translational Research Advisory Committee

UCSF Department of Neurology

2002-03	Search Committee, Academic Faculty in Epilepsy
2004-05	Search Committee, In-Residence Position in Dementia Research
2007-08	Search Committee, Academic Faculty for Alcohol Research
2007-08	Search Committee, Academic Faculty for Dementia Research
2009	Search Committee, In-Residence Position for Dementia Research
2010	Search Committee, Endowed Chair in Dementia Research
2011	Search Committee, In-Residence Position for Neurobehavioral Research

UCSF Department of Epidemiology and Biostatistics

2000	Search Committee, In-Residence Appointments
2006	Roadmap K12 Applications Committee
2010-2011	Search Committee, In-Residence Appointments

San Francisco VA Medical Center

1998-02	Clinical Research Committee, San Francisco VA Medical Center
2000-01	Chair, Search Committee, Geropsychologist, San Francisco VA Medical Center
2000-02	Search Committee, Chief of Neurology, San Francisco VA Medical Center
2004-05	Search Committee, CEO of Northern California Institute for Research and Education
2004-06	Search Committee, Geropsychiatrist
2006-08	Task Force for Geriatric Mental Health
2007-08	Search Committee, Geropsychologist
2007-08	Chair, Search Committee for Geropsychiatrist
2008-09	Board of Directors, Northern California Institute for Research and Education
2013	Search Committee, Director of the Center for Imaging of Neurodegenerative Diseases

PUBLIC SERVICE

1993-96	Guest Speaker, California College of Arts and Crafts
1996-01	Scientific Consultant, San Francisco Exploratorium Museum
1998	Guest Speaker, Korean POW Society, San Francisco, CA
1998-	Medical Scientific Advisory Council, Bay Area Alzheimer's Association

Service to Elementary and Secondary Education

Updated 10-16-14

1996-00 Met with high school students interested in careers in science and medicine. San Francisco Exploratorium

TEACHING AND MENTORING

Formal scheduled classes for UCSF Students and Trainees

1999-09 Faculty, Training in Clinical Research Program. A seven-week course on clinical research methodology organized by the UCSF Department of Epidemiology and Biostatistics; taught 10 students, 5 hours per week for seven weeks.

2000 Faculty, Meta-analysis Workshop, Training in Clinical Research Program. A three-week workshop designed to teach the fundamentals of meta-analytic techniques; taught 15 students, 5 hours per week for three weeks.

2000- Faculty, Geriatric Psychiatry Core Curriculum; 2 lectures every 6 months for psychiatry residents

2000- Faculty, Training in Clinical Research Program, Department of Epidemiology

2003 Faculty, Evidence-based Medicine Intersession Course for medical students

2007- Faculty and Co-director, Geriatric Psychiatry Seminar

2007 Faculty, Clinical Trials Course; led a section for this course

Clinical Teaching to Residents and Fellows

1995-97 Supervisor, UCSF psychiatry PGY2 residents and 3rd year medical students in neuropsychiatry consultation, San Francisco VA Medical Center

1996- Supervisor, UCSF neurology residents in the Memory Disorder Clinic, San Francisco VA Medical Center; 4 hours per week.

1997-07 Supervisor, UCSF psychiatry residents in the Memory Disorder Clinic, San Francisco VA Medical Center; 5 hours per week.

1999- Supervisor, UCSF Geriatric Fellows in Geropsychiatry and Neurology; 4 hours per week for selected month rotations

1999- Supervisor, UCSF Geropsychiatry Fellows in Geropsychiatry and Neurology; 4 hours per week for selected month rotations

2003- Training Director, Geropsychiatry at SF VAMC; supervise 1-2 geropsychiatry fellows for 6 month rotation at SF VAMC

Postgraduate Courses

2000-02 Continuing Education Course "Evidence-based Medicine"; was small group section leader of this two-day CME course for faculty on evidence-based medicine.

2000-04 Continuing Education Course "Psychiatry for Primary Care" Taught geriatric psychiatry section with 1-2 lectures per course

2004 Continuing Education Course "Geriatrics" Taught lecture on dementia treatment

2004-09 Continuing Education Course "Updates in Geriatric Psychiatry"

2006 Continuing Education Course "Women's Health"

Informal Teaching

1996- Director, Memory Disorders Clinic, San Francisco VA Medical Center; I see my own patients, as well as supervise and teach 1 nurse practitioner and 2-3 residents and 2-3 fellows who see patients in the Memory Disorders Clinic. I also lead a weekly didactic conference and a larger monthly conference on related topics.

2004- Professors Rounds, UCSF Department of Neurology: 2 hours per month

Predoctoral Students Mentored

1998-09	Isabella Fernandez, Mentor and Supervisor, MD, Senior Resident in Geriatric Psychiatry, 2 hours per week
1999-00	Rachel Whitmer, Member of Dissertation Committee, Doctor of Public Health Candidate at University of California, Davis
1999-00	Heather Bornfeld, Member of Dissertation Committee, PhD Candidate at California School of Professional Psychology
2000-01	Deborah Barnes, PhD Candidate in Epidemiology, UC Berkeley, 1 hour per week
2000-03	Nhiha Trinh, MPH, Masters in Public Health Thesis and UCSF Medical Student, 1 hour per week
2004-05	Emily Edwards, Student in Masters of Public Health, St. John's Medical School
2004-05	Cynthia Resendez, Psychiatry Resident with Geropsychiatry elective, 2 hours per month
2004-06	Karen Holden, Psychiatry Resident with Clinical Research elective, 1 hour per week
2007-09	Andrea Weinstein, Mentor and Supervisor, BA Neuropsychology, 2 hours per week
2008-10	Andrea Weston, Mentor and Supervisor, BS Brain Behavior and Cognitive Science, MPH Epidemiology, 2 hours per week
2009-10	Shawn Song, Mentor and Member of Master's Committee, UCSF Medical Student
2011-13	Alain Koyama, Mentor and Supervisor, BS Cognitive Science, MS Epidemiology, 2 hours per week
2010-13	Andrea Metti, PhD Candidate in Epidemiology, University of Pittsburgh, 2 hours per month
2010-	Tina Hoang, Mentor and Supervisor, BS Cell and Molecular Biology, MSPH Epidemiology, 2 hours per week
2010-	Cherie Vitartas, Mentor and Supervisor, BS Psychology, MPH Social and Behavioral Sciences, 2 hours per week
2011-13	Elizabeth Rose Mayeda, PhD Candidate in Epidemiology, UCSF, 2 hours per month
2012	Leah Malhotra, MD, UCSF Psychiatry Resident, 2 hours per week

Postdoctoral Students Mentored

1999-03	Kala Mehta, PhD, UCSF Geriatric Research Fellow in Geriatric Psychiatry & Epidemiology, 3 hours per week
2001-02	Rachel Whitmer, PhD, UCSF Epidemiology Postdoctoral Fellow, 2 hours per week
2002-03	Jake Elkins, MD, UCSF Neurology Fellow, 2 hours per week
2002-03	Manjula Kurella-Tamura, MD, UCSF Fellow in Nephrology, 2 hours per week
2002-04	Kaycee Sink, MD, UCSF Geriatrics Research Fellow, 2 hours per week
2002-04	Sharon Smart, MD, UCSF Biological Psychiatry Fellow, 2 hours per week
2002-05	Deborah Barnes, PhD, UCSF Research Fellow in Geriatric Epidemiology, 2 hours per week
2003-05	Rebecca Sudore, MD, UCSF Geriatrics Research Fellow, 2 hours per week
2004-05	Cynthia Resendez, MD, UCSF Psychiatry Fellow, 2 hours per week
2004-05	David Chou, MD, UCSF Geropsychiatry Fellow, 2 hours per week
2005-06	Arnaldo Moreno, MD, UCSF Geropsychiatry Fellow, 2 hours per week
2005-07	Alison Huang, MD, UCSF Internal Medicine Fellow, 2 hours per week
2006-07	Karen Holden, MD, UCSF Geropsychiatry Fellow, 2 hours per week
2006-07	Kyle Steinman, MD, UCSF Behavioral Neurology Fellow, 1 hour per month
2006-08	Adam Spira, PhD, Geriatrics Research Fellow, 2 hours per week

2007-08 Ioana Popescu, MD, UCSF Geropsychiatry Fellow, 1 hour per week
 2008-09 Alicia Romeo, MD, UCSF Geropsychiatry Fellow, 1 hour per week
 2008-10 Alexandra Fiocco, PhD, Geropsychiatry Research Fellow, 2 hours per week
 2009-10 Julia Lesselyong, PsyD, Psychiatry Research Fellow, 2 hours per week
 2009-2012 Laura Middleton, PhD, UCSF Research Research Fellow, 2 hours per week
 2010-2011 Pei-Huey Nie, MD, UCSF Geropsychiatry Fellow, 1 hour per week
 2010-2011 Kristin Krueger, PhD, Geropsychology Research Fellow, 1 hour per week
 2010-2011 Alex Threlfall, MD, UCSF Geropsychiatry Fellow, 1 hour per week
 2010-2013 Adina Zeki Al Hazzouri, PhD, UCSF Geriatric Epidemiology Fellow, 2 hours per week
 2012- Brianne Bettcher, PhD, UCSF Neuropsychology Fellow, 2 hours per week
 2012- Allison Kaup, PhD, MIRECC Fellow, 2 hours per week
 2012-14 Jennifer Yokoyama, PhD, UCSF Research fellow, 2 hours per week
 2012-13 Sophia Wang, MD, Geropsychiatry Fellow, 2 hours per week
 2013- Jasmine Nettiksimmons, PhD, Research Fellow, 2 hours per week
 2013- Raquel Gardner, MD, MIRECC Fellow, 2 hours per week
 2013- Alexis Armenakis MD, UCSF Geropsychiatry Fellow, 1 hour per week

Faculty Mentoring

2002- Cynthia Barton, NP, Assistant Clinical Professor, Department of Nursing
 2002- Rachel Whitmer, PhD, Scientist, Division of Research, Kaiser Foundation
 2003-11 Kala Mehta, PhD, Assistant Adjunct Professor, Division of Geriatrics
 2003-07 Jake Elkins, MD, Assistant Professor, Department of Neurology, Master's Committee
 2003-10 Julene Johnson, PhD, Assistant Professor, Department of Neurology
 2004-08 Manjula K. Tamura, MD, Assistant Professor, Department of Medicine, UCSF
 2005-06 Rebecca Sudore MD, Assistant Professor, Department of Medicine (Geriatrics)
 2005- Deborah Barnes, PhD, Associate Professor, Department of Psychiatry
 2006-07 Narissa Ko, MD, Assistant Professor, Department of Neurology, Master's Committee
 2007-08 Hannah C. Glass, MDCM, FRCP(C), Clinical Instructor, Department of Neurology, Master's Committee
 2007-08 Kyle Steinman, MD, Clinical Instructor, Department of Neurology, Master's Committee
 2008- Adam Spira, PhD, Assistant Professor, Department of Mental Health, Johns Hopkins Bloomberg School of Public Health
 2009- Amy Byers, PhD, Assistant Adjunct Professor, Department of Psychiatry, UCSF
 2009- Manjula K. Tamura, MD, Assistant Professor, Department of Medicine, Stanford University
 2013- Adina Zeki Al Hazzouri, PhD, Assistant Professor, Department of Epidemiology & Public Health, University of Miami
 2014- Jennifer Yokoyama, PhD, Assistant Professor, Department of Neurology, UCSF

RESEARCH AWARDS AND GRANTS

Current Research Support

1. R01 DK069406 (Multiple PI: Yaffe and Kurella)	09/05-05/15
NIDDK	\$584,439 directs/yr 1
Cognitive Decline in Chronic Renal Insufficiency	\$2,867,726 yrs 7-10; \$4,880,320 directs/total

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2. R01 AG026720 (Multiple PI: Stone and Yaffe) 07/06-03/17
 NIA \$499,990 directs/yr 1
 Change in Sleep & Cognition in Older Women \$1,999,960 yrs 6-10; \$2,319,700 directs/total

3. K24 AG031155 (PI) 09/07-05/19
 NIA \$147,425 directs/yr 1
 Predictors of cognitive aging across the lifecourse \$796,095 yrs 6-10; \$1,441,697 directs/total

4. IIRG-08-88872 (PI) 03/09-02/15
 Alzheimer's Association \$72,137 directs/yr 1
 Predictors of MCI/Dementia among the Oldest Old Women \$218,181 directs/yr 1-3 NCE

5. P50 AG023501 (Co-Investigator, PI: Miller) 04/14-03/19
 NIA \$1,384,385 directs/yr 1
 Alzheimer's Disease Research Centers: New Approaches to Dementia \$ 6,947,565 directs/yr 1-5
 Heterogeneity

6. P50 AG023501 (PI) 05/09-04/19
 NIA \$110,000 directs/yr 1
 Alzheimer's Disease Research Centers: Data and Statistical Core \$584,006 directs/yr 1-5

7. Director 07/09-06/16
 Sierra-Pacific VISN
 Mental Illness Research, Educational and Clinical Center Psychiatric Fellowship \$273,020 directs/yr 1-7

8. R01 MH086498 (PI) 03/10-02/15
 NIMH \$268,530 directs/yr 1
 Long Term Depressive Symptom Course & Adverse Health Outcomes among Older Women \$806,135 directs/yr 1-3 NCE

9. PI 04/10-11/14
 Bright Focus Foundation \$129,922 directs/yr 1
 Glucose Regulation, Cognitive & Brain Changes in Elders \$394,755 directs/yr 1-3 NCE

10. R01 AG05407 (Multiple PI: Yaffe and Cummings) 09/11-06/16
 NIA \$726,879 directs/yr 1
 Study of Osteoporotic Fractures: Successful Aging \$3,706,144 directs/yr 1-5

11. PI of Geropsychiatry Research at SF VA Medical Center 11/11-10/16
 Sierra-Pacific VISN
 Mental Illness Research, Educational and Clinical Center \$1,150,000 directs/yr 6-10

12. W81XWH-11-2-0189 (Co-investigator, PI: Byers) 01/12-12/14
 Department of Defense \$124,953 directs/yr 1
 Late Life PTSD and Adverse Health Outcomes among U.S. Veterans \$249,906 directs/yr 1-2

13. R01 MD007019 (Co-investigator, PI: Byers) 04/12-12/15

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NIH: National Institute on Minority Health and Health Disparities	\$200,000 directs/yr 1
Epidemiology of Suicidal Behavior in Racially/Ethnically Diverse	\$800,000 directs/yr 1-4
Older Americans	

14. 2P01 AG019724 (PI: Miller; Core PI: Yaffe)	09/12 – 08/17
NIA	\$123,833 directs/yr 1
Frontotemporal Dementia: Genes, Images, and Emotions: Data	\$619,165 directs/yr 1-5
Management and Biostatistics Core	

15. W81XWH-12-1-0581 (PI: Yaffe)	10/12 – 03/15
Department of Defense	\$274,955 directs/yr 1
Endophenotypes of Dementia Associated with Traumatic Brain Injury	\$446,187 directs/yr 1-2
in Retired Military Personnel	

16. P30 AG044281 (Co-Investigator, PI: Covinsky)	07/13-06/18
NIA	\$682,650 directs/yr 1
UCSF Older American Independence Center	\$3,413,250 directs/ yrs 1-5

17. (Co-Investigator, PI: Cifu)	07/13-06/18
Department of Defense/VA	
Chronic Effects of Neurotrauma Consortium (Military and Veterans	\$62,500,000 directs/ yrs 1-5
Rehabilitation and Recovery from Injury Network)	

18. 81XWH-13 (PI: Yaffe)	10/14-09/17
Department of Defense	\$199,458 directs/yr 1
Blood biomarker profile of TBI-associated cognitive impairment	\$593,534directs/ yrs 1-3
among old & young veterans	

Pending Research Support

1. (PI: Yaffe)	10/14-05/18
Department of Defense/VA	
Epidemiology Project. Chronic Effects of Neurotrauma Consortium (Military and Veterans	
Rehabilitation and Recovery from Injury Network, Notified of plans for funding)	

2. (Multiple PI: Yaffe and Sidney)	12/14-11/18
NIA	
Determinants of Midlife & Longitudinal Change in Cognitive Function: CARDIA Study (Received	
5 th percentile, awaiting council review)	

Mentored Career Development Awards

1. Career Development Award (Co-Mentor, PI: Manjula Kurella)	07/04-06/06
American Society for Nephrology	
Cognitive Impairment in Elderly Persons with Chronic Kidney Disease	

2. K23 NS046302 (Co-Mentor, PI: Jake Elkins)	07/04-08/07
NINDS	
Hypertension and High Cognitive Function in the Elderly	

3. K01 DK066308 Award (Primary Mentor, PI: Rachel Whitmer) 10/05-09/09
NIDDK
Diabetes and Cognitive Function
4. K01 AG024069 (Primary Mentor, PI: Deborah Barnes) 10/05-09/10
NIA
Physical Activity and Cognitive Function
5. K01 AG025444 (Co-Mentor, PI: Kala Mehta) 10/05-09/11
NIA
Race/Ethnicity, Cognitive Function and Health Outcomes
6. K01 MH079093 (Primary Mentor, PI: Amy Byers) 01/07-12/11
NIMH
The Complexity of Late Life Depression: Bridging Methods and Clinical Substance
7. Paul B. Beeson Career Development Award (Primary Mentor, PI: Manjula Kurella) 09/07-08/12
NIA
Mechanisms of Cognitive Impairment in Chronic Kidney Disease
8. Canadian Institutes of Health Research Fellowship (Primary Mentor, PI: Alexandra Fiocco) 07/08-06/11
Canadian Institutes of Health Research
Biomarkers of Optimal Cognitive Aging
9. Canadian Institutes of Health Research Fellowship (Primary Mentor, PI: Laura Middleton) 07/09-07/12
Canadian Institutes of Health Research
Predictors of Healthy Cognitive Aging in the Oldest Old
10. K01 AG033195 (Co-Mentor, PI: Adam Spira) 12/09-11/14
NIA
Sleep Disturbances and Functional Decline in Elders
11. Postdoctoral Fellowship (Primary Mentor, PI: Adina Zeki Al Hazzouri) 01/12-12/13
American Academy of Neurology Foundation/American Heart Association/ American Stroke Association
Cardiovascular risk factors for stroke and consequences of stroke among three racial/ethnic groups
12. K23 AG042492 (Co-Mentor, PI: Brianne Bettcher) 08/12-07/17
NIA
Cognitive and Neural Correlates of Inflammation in Healthy Older Adults
13. K23 AG040772 (Co-Mentor, PI: Alexander Smith) 09/12-08/17
NIA
Late Life Disability: Epidemiology, Symptoms, Quality of Life Duration

13. KL2 (Primary Mentor, PI: Adina Zeki Al Hazzouri) 10/13-04/14
Clinical and Translational Science Institute (CTSI)
Effects of race and lifecourse cardiovascular risk on neuropsychiatric outcomes
14. K01 AG047273 (Primary Mentor, PI: Adina Zeki Al Hazzouri) 04/14-03/19
NIH/NIA
Lifecourse cardiovascular risk, depression and cognition in black & white adults
15. K01 (Co-Mentor, PI: Jennifer Yokoyama) 09/14-08/19
NIH/NIA
RNA signatures of frontotemporal dementia and ALS due to C90RF72 expansion
16. Career Development Award (Primary Mentor, PI: Allison Kaup) 12/14-11/19
Veterans Health Administration
Cognitive Reserve and Training in Older Veterans with Traumatic Brain Injury

Past Research Support

1. PI of the Investigator-initiated Dementia Study 07/95-06/02
Lilly Laboratories \$342,152 directs/yr 1
Multicenter Osteoporosis Raloxifene Evaluation \$2,395,066 directs/yr 1-7
2. Co-investigator, PI Wolkowitz 10/95-10/97
National Institute on Aging STTR \$50,000 directs/yr 1
Dehydroepiandrosterone Treatment of Alzheimer's Disease \$100,000 directs/yr 1-2
3. IIRG 95-174 (Co-investigator, PI Wolkowitz) 10/95-09/99
Alzheimer's Association \$37,209 directs/yr 1
Longitudinal Study of Adrenocorticoids and Functioning in Alzheimer's Disease \$148,835 directs/yr 1-4
4. PI 05/97-11/97
National Institute on Aging Contract
Bone Mineral Density and Cognitive Decline \$4,500 directs/yr 1
5. Project Director of the Cognitive Substudy 07/97-12/98
Wyeth-Ayerst
The Heart & Estrogen-Progestin Replacement Study \$196,000 directs/yr 1
6. Young Investigator Award (PI) 07/97- 06/00
NARSAD \$20,000 directs/yr 1
Depression and Cognitive Decline in Elderly Women \$60,000 directs/yr 1-3
7. 1K23 AG00888 (PI) 09/98-08/04
NIA \$93,958 directs/yr 1
Estrogenic Compounds, Cognition and Dementia in Older Women \$563,750 directs/yr 1-6

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8. PI of Cognitive Substudy Berlex Labs Ultra Low-dose Transdermal Estrogen Replacement Assessment (ULTRA)	07/99-06/03 \$587,500 directs/yr 1 \$2,350,000 directs/yr 1-4
9. PI of UCSF site NIA Alzheimer's Disease Cooperative Study	02/99-12/03 \$21,450 directs/yr 1 \$85,800 directs/yr 1-4
10. PI Pfizer Inc The Effect of Lasofoxifene on Cognitive Function	07/00-12/03 \$51,187 directs/yr 1 \$153,562 directs/yr 1-3
11. Young Investigator Award (PI) NARSAD Outcomes of Depression in Multi-Ethnic Frail Elders	07/01-12/04 \$20,000 directs/yr 1 \$60,000 directs/yr 1-3
12. Beeson Faculty Scholars Program (PI) American Federation of Aging Research The Determinants of Cognitive Change and Outcomes in African-American and White Elders	07/01-06/06 \$90,000 directs/yr 1 \$450,000 directs/yr 1-5
13. PI Wyeth Ayerst Cognitive Complaints in Early Menopause Estrogen Trial (COGENT)	01/02-12/04 \$27,500 directs/yr 1 \$55,000 directs/yr 1-2
14. D01 HP 00015-02 (Co-investigator, PI: Landefeld) HRSA Geriatric Training for Physicians, Dentists, and Behavioral & Mental Health Professionals	07/02-06/06 \$623,737 directs/yr 1 \$2,494,949 directs/yr 1-4
15. PI UCSF/Mt Zion Women's Health Clinical Research Center Identification of Modifiable Risk Factors for Cognitive Impairment among Older Women	02/03- 02/05 \$20,000 directs/yr 1 \$40,000 directs/yr 1-2
16. R01 AG010897 (Co-Investigator, PI: Weiner) NIA Prediction of Cognitive Decline with MRI/MRS	04/03-12/08 \$646,110 directs/yr 1 \$3,230,550 directs/yr 1-5
17. K01 HP 00066-01 (PI) HRSA Geriatric Academic Career Award	09/03-09/06 \$55,000 directs/yr 1 \$165,000 directs/yr 1-3
18. R01 AG021918 (PI) NIA Sex Hormones, Related Polymorphisms & Cognitive Decline	09/03-08/09 \$224,726 directs/yr 1 \$881,992 directs/yr 1-6

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19. R01 AG005407 (Co-investigator, PI: Black)	11/03-06/11
NIA	\$197,368 directs/yr 1
Study of Osteoporotic Fractures	\$1,578,940 directs/yr 1-8
20. P50 AG023501 (Co-Investigator and Co-Director of Clinical Core, PI: Miller)	04/14-03/19
NIA	\$1,400,000 directs/yr 1
Alzheimer's Disease Research Centers	\$7,000,000 directs/yr 1-5
21. Independent Investigator Award (PI)	09/04-09/08
NARSAD	\$33,000 directs/yr 1
Depressive Symptoms and Risk of Mild Cognitive Impairment: The Role of Inflammation and Cerebrovascular Disease	\$100,000 directs/yr 1-4
22. PI	06/05-04/09
Organon, Inc	\$20,767 directs/yr 1
LIFT Trial Ancillary Study	\$83,071 directs/yr 1-4
23. R25 MH060482 (Co-Investigator, PI Reus)	07/05-06/09
NIMH	\$62,500 directs/yr 1
Training Next Generation of Mental Health Researchers	\$250,000 directs/yr 1-4
24. R21 DK070713 (PI)	09/05-08/07
NIDDK	\$100,000 directs/yr 1
Metabolic Syndrome and Cognitive Decline	\$200,000 directs/yr 1-2
25. Co-Investigator, PI: Barnes	04/07-03/10
Alzheimer's Association	\$80,000 directs/yr 1
Computer-Based Cognitive Training in Older Adults with Memory Complaints	\$240,000 directs/yr 1-3
26. W81XWH-05-2-0094 (PI)	08/08-03/11
Department of Defense	\$140,847 directs/yr 1
Post Traumatic Stress Disorder, Traumatic Brain Injury and Risk of Dementia	\$281,694 directs/yr 1-3
27. TL1 RR024129 (Co-Investigator, PI: SC Johnston)	09/06-08/11
NIH: National Center for Research Resources	\$ 1,091,667 directs/yr 1
Clinical and Translational Science Institute	\$6,550,000 directs/yr 1-5
28. PI	09/06-03/12
Anonymous foundation support	\$154,789 directs/yr 1
Biomarkers of Optimal Cognitive Aging	\$415,407 directs/yr 1-5
29. R03 AG033751 (Co-investigator, PI: Haan)	06/10-06/12
NIA	\$41,000 directs/yr 1
Epidemiology of Hypertension and Obesity in Older Mexican Americans	\$82,000 directs/yr 1-2

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30. R01 AG028481 (Co-Investigator, PI: Covinsky)	09/07-10/12
NIA	\$150,00 directs/yr 1
Depressive Symptoms, Aging, Disability and Health Outcomes	\$600,000 directs/yr 1-4
31. R01 AG010897 (Co-investigator, PI: Weiner)	04/09-03/14
NIA	\$483,407 directs/yr 1
Prediction of cognitive decline with structural, diffusion, and perfusion MRI	\$2,417,035 directs/yr 1-5
32. 15945 (Co-Investigator of the Center and Director of the Translational Core, PI: H Rosen)	07/12-06/15
California Department of Public Health	\$300,000 directs/yr 1
UCSF Alzheimer's Disease Research Center of California	\$900,000 directs/yr 1-3

INVITED PRESENTATIONS

International

Hormones and Dementia. Women's Cognitive and Emotional Health Conference, Northampton, England, September 1999.

Effect of Raloxifene on the Prevention of Dementia and Cognitive Impairment in Older Women. International Conference on Alzheimer's Disease and Related Disorders, Stockholm, Sweden, July 2002.

Genetics and Cognition. International Society for Menopause, Pisa, Italy, March 2003.

Glycosylated Hemoglobin Level and Development of Cognitive Impairment in Older Women. International Conference on Alzheimer's Disease and Related Disorders, Philadelphia, PA, July 2004.

Endogeneous & Exogeneous Estrogens. International Conference on Alzheimer's Disease and Related Disorders, Madrid, Spain, July 2006.

PPAR γ genotype and Risk of Cognitive Impairment in Elders. International Conference on Alzheimer's Disease and Related Disorders, Madrid, Spain, July 2006.

Effects of Ultra Low-dose Transdermal Estradiol on Cognition and Health-Related Quality of Life. International Conference on Alzheimer's Disease and Related Disorders, Madrid, Spain, July 2006.

Prevention of Dementia. Canadian Colloquium of Dementia (Plenary Address), Vancouver, Canada, October 2007.

Insulin Resistance/Metabolic Syndrome and Cognitive Impairment (Session Chair and Presenter). International Conference on Alzheimer's Disease and Related Disorders, Chicago, IL, July 2008.

Impact of Maintaining Cognitive Function on Physical Disability and Death in a Biracial Cohort of Older Women and Men. International Conference on Alzheimer's Disease and Related Disorders, Chicago, IL, July 2008.

Telomere Length and Cognitive Function in Community-Dwelling Elders: Findings from the Health ABC study. International Conference on Alzheimer's Disease and Related Disorders, Chicago, IL, July 2008.

Post-traumatic Stress Disorder and Risk of Dementia among U.S. Veterans. International Conference on Alzheimer's Disease and Related Disorders, Vienna, Austria, July 2009.

Chronic Kidney Disease and Cognitive Function in Older Adults: The CRIC Cognitive Study. International Conference on Alzheimer's Disease and Related Disorders, Vienna, Austria, July 2009.

Strategies to Prevent Dementia: The Importance of Lifestyle Factors (Plenary Address). The 52nd Annual Meeting of the Japan Geriatrics Society, Kobe, Japan, June 2010.

What Factors Might Prevent Dementia (Plenary Address). International Conference on Alzheimer's Disease and Related Disorders, Honolulu, HI, July 2010.

The Neuropathological Features Associated with Alzheimer's Disease in the Oldest Old Versus the Young Old. International Conference on Alzheimer's Disease and Related Disorders, Honolulu, HI, July 2010.

Objectively Measured Sleep Quality and Risk of Cognitive Impairment. International Conference on Alzheimer's Disease and Related Disorders, Paris, France, July 2011.

Sleep Disorders and Cognitive Function in Older Women. International Conference on Alzheimer's Disease and Related Disorders, Vancouver, Canada, July 2012.

Cardiovascular risk factors for cognitive function: effects from early adulthood to mid-life. International Conference on Alzheimer's Disease and Related Disorders, Boston, MA, July 2013.

Maximizing Cognitive Health (Keynote Lecture Series). Dementia Awareness Week, Brain Health: Making the Connections, Sydney, Darwin, Alice Springs, Canberra, Melbourne, Adelaide, Perth and Brisbane, Australia, September 2013.

Lifestyle Issues for Prevention. Dementia Collaborative Research Centres Forum, Brisbane, Australia, September 2013.

Improving Life and Care for People Affected by Dementia, and their Carers (Invited Scientist). G8 Dementia Summit, London, United Kingdom, December 2013.

Risk factors for cognitive impairment: need for a lifecourse approach. Council of the German Center for Neurodegenerative Diseases (DZNE) Senate Meeting, Bonn, Germany, May 2014.

The Importance of Risk Factor Modification over the Lifecourse. International Conference on Alzheimer's Disease and Related Disorders, Copenhagen, Denmark, July 2014.

Late-life Psychiatric Disease as a Risk Factor for Neurodegenerative Disease. Visiting Professor, Centre for Addiction and Mental Health, Toronto, Canada, October 2014.

National

Apolipoprotein E Phenotype and Mild Cognitive Decline in Elderly Women. American Academy of Neurology Annual Meeting, Boston, MA, April 1997.

Estrogen and Alzheimer's Disease. The French Foundation for Alzheimer's Research Annual Meeting, Los Angeles, CA, February 1998.

Depressive Symptoms and Cognitive Decline in Nondemented Elderly Women. Presented as the Young Investigator Award Lecture at the American Association of Geriatric Psychiatry Annual Meeting, New Orleans, LA, March 1999.

Estrogen, Cognitive Function and Dementia. Symposium for Women's Issues in Geriatric Psychiatry, Miami, FL, March 2000.

Selective Estrogen Receptor Modulators and Dementia: What is the Evidence? The NIH Workshop on Selective Estrogen Receptor Modulators, Bethesda, MD, April 2000.

Effects of Raloxifene on Cognitive Function in Postmenopausal Women without Dementia. American Academy of Neurology, San Diego, CA, April 2000.

Epidemiology and Treatment Trials of Estrogen for Alzheimer's Disease. World Alzheimer's Congress, Washington, DC, July 2000.

Prevention of Alzheimer's Disease. The Langley Seminar, Legacy Health Systems, Portland, OR, February 2001.

Pharmacological Interventions to Treat or Prevent Dementia. Stanford University and Palo Alto VA Medical Center Third Annual Update on Dementia Conference, Palo Alto, CA, March 2001.

Serum Lipoprotein Levels, Statin Use, and Cognitive Function in Older Women. American Academy of Neurology, Philadelphia, PA, May 2001.

Walking Protects Women From Cognitive Decline. American Academy of Neurology, Philadelphia, PA, May 2001.

Prevention of Dementia. Wake-Forest University, Sticht Center of Aging Research Conference, Winston-Salem, NC, October 2001.

Inflammatory Markers and Cognitive Decline in Well-Functioning Elders. American Academy of Neurology, Denver, CO, April 2002.

Predictors of Nursing Home Placement in Patients with Dementia: Importance of Both Patient and Caregiver Characteristics. American Academy of Neurology, Denver, CO, April 2002.

Modifiable Risk Factors for Alzheimer's Disease. Society for Epidemiological Research, Palm Springs, CA, June 2002.

Treatment and Prevention of Dementia, Harvard Division of Geriatrics, Boston, MA, November 2003.

A Randomized, Controlled Trial of Antioxidants and Zinc and the Impact on Cognition in the Elderly. American Neurological Association, San Francisco, CA, November 2003.

Antioxidants and Prevention of Cognitive Decline, American Society for Geriatric Psychiatry. Baltimore, MD, February 2004.

Metabolic Syndrome and Cognitive Decline: The Effect of Inflammation. American Academy of Neurology, San Francisco, CA, April 2004.

Prevention of Alzheimer's Disease. World Federation of Neuroepidemiology Keynote Presentation, Oakland, CA, May 2004.

Update on the Role of Hormones and Dementia (Plenary Address). American Society for Geriatric Psychiatry. San Diego, CA, February 2005.

The Role of Hormones in Mental Health (Plenary Address). American Society for Geriatric Psychiatry, San Diego, CA, March 2005.

Lasofloxifene Does Not Affect Cognitive Function in Postmenopausal Women: Results From Two Phase-3 Randomized Controlled Clinical Trials for Osteoporosis Prevention. American Academy of Neurology, Miami, FL, April 2005.

Subtype of Mild Cognitive Impairment and Progression to Dementia and Death. American Academy of Neurology, Miami, FL, April 2005.

Modifiable Risk Factors for Cognitive Decline (Plenary Address). North American Menopause Society, San Diego, CA, September 2005.

The Role of Biomedical Factors in Explaining Socio-Economic Differences in Cognitive Decline. Gerontological Society of America, Orlando, FL, November 2005.

Cognitive Function in Chronic Kidney Disease. NIDDK Kidney Interagency Coordinating Subcommittee Meeting, Bethesda, MD, March 2006.

Cognitive Decline is Associated with Sleep Disturbance in Older Community-Dwelling Women. American Academy of Neurology, San Diego, CA, April 2006.

Endogenous Sex Hormone Levels and Risk of Cognitive Decline in a Biracial Older Cohort. American Academy of Neurology, San Diego, CA, April 2006.

Metabolic Syndrome and Dementia. NIA Obesity and Alzheimer's Disease Workshop, Bethesda, MD, June 2006.

Visiting Professor. Johns Hopkins Bloomberg School of Public Health, December 2006.

Visiting Scholar. Institute on Aging, University of Pennsylvania School of Medicine, April 2007.

Metabolic Syndrome and Cognitive Decline in Elderly Latinos. American Academy of Neurology, Boston, MA, May 2007.

Estrogen receptor genotype and risk of cognitive impairment in elders: Findings from the Health ABC study, American Academy of Neurology, Boston, MA, May 2007.

Cognitive Aging Summit, NIA, Washington DC, October, 2007.

Prevention of Dementia (Plenary Address). American Academy of Neurology Annual Meeting, Chicago, IL, April 2008.

Cystatin-C is a Marker for Cognitive Function: Findings from the Health ABC Study. American Academy of Neurology Annual Meeting, Chicago, IL, April 2008.

Predictors & Outcomes of Maintaining Cognitive Function: A Model of Successful Aging. Fidelity Foundation Conference, Bar Harbor, ME, August 2008.

NIA-Alzheimer's Association Conference on Alzheimer's Disease Prevalence. Washington DC, March, 2009.

Subgroups at Risk for Dementia. Alzheimer's Association Roundtable. Washington DC, October, 2010.

Dementia Prevention (Keynote Address). West Coast Geriatric Psychiatry Conference, San Diego, CA, February 2011.

Special Populations for Cognitive Research: the Oldest Old and Successful Aging. Women's Health Initiative Steering Committee Meeting, Seattle, WA, May 2011.

Alzheimer's Epidemiological Perspective (Keynote Address). Alzheimer's Association Roundtable. Washington DC, October 2011.

Prevention of Alzheimer's Disease: Dashed Hopes and Future Promises (Keynote Address). Alzheimer's Association Meeting of the Minds Dementia Conference. St. Paul, MN, March 2012.

Sleep Quality and Cognitive Impairment. (Visiting Professor) Mayo Clinic Department of Neurology. Rochester, MN, March 2012.

Why Aging Veterans May be at Risk for Dementia. Alzheimer's Association: Military Risk Factors for Alzheimer's Disease. Arlington, VA, May, 2012.

Objectively measured sleep and risk of dementia. Workshop on AD and Related Disorders. Napa, CA, July 2012.

Non-pharmacological Strategies for Prevention of Alzheimer's Disease. 22nd Annual Matthew & Marcia Simons Research Symposium on Alzheimer's Disease. Newton, MA, November 2012.

Long-term depressive symptom burden and risk of cognitive decline and dementia among very old women. American Association for Geriatric Psychiatry, Los Angeles, CA, March 2013.

Lifestyle interventions for dementia and its prevention. (John Mackey Award Lecture). 19th Annual Update on Alzheimer's and Related Dementias: Defining the Standard of Care. Baltimore, MD, April 2013.

Projecting the Effect of Dementia Risk Factor Reduction on Alzheimer's Disease Prevalence. Translation of Available Scientific Evidence into Primary Prevention of Alzheimer's Disease and Cognitive Aging. New York, NY, April 2013.

The Long-term Course and Effects of Depressive Symptoms in Older Women. American Association for Geriatric Psychiatry Annual Meeting. Orlando, FL, March 2014.

Modifiable Risk Factors for Cognitive Aging: Lessons Learned (Distinguished Scientist Award). American Association for Geriatric Psychiatry Annual Meeting. Orlando, FL, March 2014.

Prevention: An Overview. Alzheimer's Disease and Related Dementias: Updates in Prevention, Treatment and Innovative Care. San Diego, CA, May 2014.

Lifestyle Interventions. Alzheimer's Disease and Related Dementias: Updates in Prevention, Treatment and Innovative Care. San Diego, CA, May 2014.

Long-Term Effects of Traumatic Brain Injury. The Brain at War Conference. San Francisco, CA, October 2014.

Regional

Epilepsy and Pregnancy. UCSF Department of Neurology Grand Rounds, San Francisco, CA, 1993.

Current Topics in Epilepsy. Northern California Epilepsy Society, San Francisco, CA, 1993.

Apolipoprotein E and Dementia. UCSF Department of Psychiatry Grand Rounds, San Francisco, CA, October 1996.

Endogenous and Exogenous Estrogen in Postmenopausal Women: Effects on Cognition and Dementia. Columbia University Sergievsky Center Seminar. New York, NY, February 1997.

Prevention and Treatment of Alzheimer's Disease. The UCSF National Center of Excellence in Women's Health Symposium, "Alzheimer's Disease and Women: From the Bench to the Bedside and Beyond," San Francisco, CA, March 1998.

Estrogen, Cognition and Dementia. UCSF Department of Neurology Grand Rounds, San Francisco, CA, November 1998.

Women's Health Research. UC Davis Women's Health Conference, Davis, CA, November 1998.

Estrogen and Dementia. Pharmacotherapy for Perimenopausal and Menopausal Women, San Francisco, CA, September 1999.

Estrogen and Apolipoprotein E: Evidence of Gene-Environment Interaction. Northern California Epidemiology Conference, San Francisco, CA, November 1999.

Psychiatry and Neurology: Neural Notations. Department of Psychology, University California, Santa Cruz, CA, April 2000.

Estrogens, SERMS and Dementia. Grand Rounds, UCSF Department of Obstetrics and Gynecology, San Francisco, CA, December 2000.

Estrogens, SERMS and Cognition in Older Women: What is the Evidence? Updates in Obstetrics and Gynecology, UCSF Continuing Medical Education Program, San Francisco, CA, October 2001.

Geriatrics Update: Treatment and Prevention of Alzheimer's Disease. UCSF Psychiatry for Primary Care CME Course, San Francisco, CA, April 2002.

Prevention of Dementia. UCSF Geriatrics for Primary Care CME Course, San Francisco, CA, May 2002.

UCSF Geriatric Psychiatry CME Course, San Francisco, CA, 2004.

Diagnosis of Dementia. UCSF Geriatric Psychiatry CME Course, San Francisco, CA, September 2005.

Updates on Treatment of Dementia. UCSF Women's Health CME Course, San Francisco, CA, October, 2006.

Bridging Cultures: Improving Evaluation and Treatment of Cognitive Disorders. UCSF Geriatric Psychiatry CME Course, San Francisco, CA, April 2008.

Telomere Length and Cognitive Function in Community-Dwelling Elders: Findings from the Health ABC Study (Best Oral Presentation Award). Bay Area Clinical Research Symposium, San Francisco, CA, October 2008.

The Metabolic Syndrome and Cognitive Impairment. Bay Area Clinical Research Symposium, Novato, CA, June 2009.

Prevention of dementia: What looks promising? UCSF Department of Psychiatry Grand Rounds, San Francisco, CA, May 2010.

Research Talk. San Francisco VA Medical Center, San Francisco, CA, March 2011.

Prevention of Dementia in 2011. UCSF Department of Internal Medicine Grand Rounds, San Francisco, CA, March 2011.

Prevention and Treatment of Dementia. UCSF Department of Internal Medicine CME Course, San Francisco, CA, October 2011.

Late-life Psychiatric Disease as a Risk Factor for Neurodegenerative Disease. Northern California Psychiatric Society and UCSF Memory and Aging Center CME Course, San Francisco, CA, November 2011.

Cognitive Function and Dementia in Older Women. (Keynote Address). UC Davis Women's Health Conference. Napa, CA, June 2012.

Diagnosis and Prevention of Alzheimer's Disease. Genetech. South San Francisco, CA, March 2013.

Early adult risk factors for cognitive impairment. UCSF Memory and Aging Center Grand Rounds. July, 2013.

Optimizing Cognitive Aging. UCSF Academic Senate Annual Faculty Research Award in Clinical Science. October, 2013.

Strategies for Prevention of Cognitive Aging. Stanford University Department of Neurology Grand Rounds. March, 2014.

Lifestyle Risk Factors for Dementia Prevention. Stanford University Department of Psychiatry and Behavioral Sciences Grand Rounds. September, 2014.

Recent Most Significant Publications (*Dr. Yaffe's role in italics*)

Yaffe K, Vittinghoff E, Lindquist K, Barnes DE, Covinsky KE, Neylan T, Kluse M, Marmar C. Post-traumatic stress disorder and risk of dementia among U.S. Veterans. *Archives of General Psychiatry*. 2010; 67(6):608-613. *I designed the study, obtained funding, analyzed the data, and wrote the manuscript.*

Yaffe K, Weston A, Graff-Radford NR, Satterfield S, Simonsick EM, Younkin SG, Younkin LH, Kuller L, Ayonayon HN, Ding J, Harris TB. Association of plasma beta-amyloid level and cognitive reserve with subsequent cognitive decline. *JAMA*. 2011; 305(3):261-266. *I designed the study, obtained grant funding, analyzed the data, prepared the data presentation and wrote the manuscript.*

Yaffe K, Laffan AM, Litwack Harrison S, Redline S, Spira AP, Ensrud KE, Ancoli-Israel S, Stone KL. Sleep disordered breathing, hypoxia, and risk of mild cognitive impairment & dementia in older

women. JAMA. 2011; 306(6):613-9. *I designed the study, obtained grant funding, analyzed the data, prepared the data presentation and wrote the manuscript.*

Barnes DE, **Yaffe K**. The Projected Impact of Risk Factor Reduction on Alzheimer's Disease Prevalence. The Lancet Neurology. 2011; 10(9):819-28. *I co-designed and led the study with Dr. Barnes, including obtaining grant funding, data analysis, and writing the manuscript.*

Larson EB, **Yaffe K**, Langa KM. New Insights into the Dementia Epidemic. New England Journal of Medicine. 2013; 369(24):2275-7. *I co-designed and co-wrote this manuscript.*

PEER REVIEWED PUBLICATIONS

Cited over ***300, **200, *100 times. Dr. Yaffe's h-index is 85.

1. Papiernik E, Bouyer J, **Yaffe K**, Winisdorffer G, Colin D, Dreyfus J. Women's acceptance of a preterm birth prevention program. American Journal of Obstetrics and Gynecology. 1986; 155(5):939-46.
2. ***Yaffe K**, Lowenstein, D. Prognostic factors of pentobarbital therapy for refractory generalized status epilepticus. Neurology. 1993; 43(5):895-99.
3. ***Yaffe K**, Ferriero D, Barkovich J, Rowley H. Reversible MRI abnormalities following seizures. Neurology. 1995; 45(1):104-108.
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5. *****Yaffe K**, Sawaya G, Lieberburg I, Grady D. Estrogen therapy in postmenopausal women: Effects on cognitive function and dementia. JAMA. 1998; 279(9):688-695.
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8. Dwyer E, Arean P, **Yaffe K**, Callahan C. Minor depression in the elderly. Journal of Clinical Outcomes Management. 1999; 6(2):41-51.
9. *Matthews K, Cauley J, **Yaffe K**, Zmuda J. Estrogen replacement therapy and cognitive decline in older community women. Journal of the American Geriatrics Society. 1999; 47(5):518-23.
10. ****Yaffe K**, Blackwell T, Gore R, Sands L, Reus V, Browner W. Depressive Symptoms and Cognitive decline in nondemented elderly women: A prospective study. Archives of General Psychiatry. 1999; 56(5):425-30.

11. Cauley J, Zmuda J, **Yaffe K**, Kuller L, Ferrell R, Wisniewski S, Cummings S. Apolipoprotein E polymorphism: A new genetic marker of hip fracture risk--The Study of Osteoporotic Fractures. *Journal of Bone Mineral Research*. 1999; 14(7):1175-1181.
12. Stoltz C, Baime M, **Yaffe K**. Depression in the patient with Rheumatologic Disease. *Rheumatological Disease Clinics of North America*. 1999; 25(3):687-702.
13. **Yaffe K**, Browner W, Cauley J, Launer L, Harris T. Association between bone mineral density and cognitive decline in older women. *Journal of the American Geriatrics Society*. 1999; 47(10):1176-82.
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